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Cardio-vascular Diseases

RECENT ADVANCES IN THEIR ANATOMY, PHYSIOLOGY, PATHOLOGY, DIAGNOSIS AND TREATMENT

BY

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"Progress is the Law of Life"—BROWNING.

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To
I. B. S

PREFACE.

Shortly after the appearance of my "Diseases of the Heart and Aorta," in 1905, I began to issue a series of monographs as successive addenda to the volume. They included a number of topics that were gradually and deservedly coming into prominence, or required recasting in the light of newer experience. These monographs have now been revised and are presented to the profession in book form. For this opportunity I am indebted to the courtesy of the medical journals which published them.

The purpose of my book has been to furnish intelligible and at the same time condensed accounts of these matters, rather than summaries of definite conclusions. No one can doubt that additional discoveries will be made in cardiac anatomy, physiology, pathology and therapy, calling for corresponding alterations in our present views; for cardiology, like all branches of medicine, is a progressive science. Indeed, even when we have accepted the recent findings of Keith, Flack, Tawara, and their fellow anatomists, and after fully recognizing the value of the data furnished by such physiologists as Gaskell, Einthoven, Lewis and Cushny, we should realize that other competent men will extend their work. In fact, from the reports that from time to time trickle down to us from such sources, it is evident that the stores of information in these departments are still being added to, while pathologists and therapists are even more actively cultivating their respective fields.

But even if there are depths in any branch that have not been fathomed, we should not therefore deter ourselves from gathering together what new stores we have at hand, if they can be profitably laid before the profession. Especially is this true in the matter of cardiovascular diseases, where the advances of the last seven years have been numerous and important. And when these diseases are felt to be only second to the tubercular in the calendars of fatality, almost every item bearing on them awakens a lively interest.

So much for my justification in presenting a new book. As for its particular scope, it should be premised that I have purposely avoided perplexing questions, or those of merely academic interest, and have tried to limit myself to the description of those newer

features of the subject that have a definite bearing on diagnosis and treatment.

Modern sphygmomanometry, its appliances, the methods of operating them, its utility and its limitations, have claimed a good deal of space.

Even more has been given to graphic methods and appliances, electrocardiography, phonocardiography, micrography, and other varieties of polygraphy. Those that are best adapted for laboratories and the larger hospitals have been distinguished from the class better suited for office or bedside practice. Viscicometry has also been discussed, not so much for its present value, but rather for its future possibilities.

Arrhythmias have been classified on a physiological basis, and their differentiation by graphic and other methods compared.

Other subjects included in this volume but not elaborated in the former one are the uses of carbon dioxid, mobility and malpositions of the heart, and cardiovascular thromboses. The chapters on myocardial diseases and malignant endocarditis are essentially new.

Of the illustrations only one appeared in the former volume. In fact, the majority were prepared for the author from original sources. Some, however, have been borrowed from Keith, Mackenzie, Lewis, and Wenckebach. A few have been modified. Wherever they appear in the text, an acknowledgement of their sources has been made. It has been thought desirable to illustrate each subject amply.

Wherever practicable, a special effort has been made to avoid technical phraseology, and to use simple terms, adhering as far as possible to the English system of notation. This has not always been an easy task. Physiologists, clinicians, and mechanicians are sometimes disposed to employ terms that are needlessly technical, and the two former an arbitrary system of notation in their graphic tracings. The blame is not to be charged to the profession of any one country. It should, however, be the duty of all of us who are interested in the advance of scientific medicine to deprecate the use of new words or special systems of notation, so long as there are standard words and notation systems in common use and generally understood.

It would be going too far afield for me to enumerate the numerous sources from which aid has been given me in the preparation of this volume. It has come both from within and without professional circles. But I wish here to express my thanks par-

ticularly to Dr. T. B. Barringer, Jr., and Dr. Frank S. Meara of this city; to Dr. George Bachmann of Atlanta, Georgia, and to Drs. Edmund Hoke and Theodor Wohrizek of Franzensbad, Austria. Whenever others have given me assistance, allusion has been made to it in the text.

THOMAS E. SATTERTHWAITE.

7 East Eightieth Street,
New York City.
Oct., 1912.

Cardio-vascular Diseases

Recent Advances in their Anatomy, Physiology, Pathology Diagnosis and Treatment

Chapter I.

SOME RECENT ADVANCES IN OUR KNOWLEDGE OF THE ANATOMY AND PHYSIOLOGY OF THE HEART.

During the last ten years a series of investigations has been carried on with reference to the gross and minute anatomy of the heart, the result being that some important discoveries have been made. At the same time physiologists have altered some of our former conceptions, especially as to the reciprocal relations of cardiac, arterial and venous activities. This has been accomplished by a conjoint use of some of the newer recording instruments, of which the electrocardiograph and the phonophotograph are examples.

Of the first series of investigations alluded to, the discovery of the auriculoventricular or His bundle, its point of origin and its final distribution, is the most noteworthy result. The gradual steps that led to the discovery are worthy of summarizing. In the first place, it has long been known that at an early period in embryonic life the heart is a tube, at one end of which is the *sinus venosus,* where the venous trunks unite (Fig. 1). From this tube pouches develop, to become on the one hand an auricle *B,* and on the other a ventricle *A,* while the original tube still connects them. Later, in connection with the sinus are formed the superior vena cava *H* and the inferior vena cava *F,* a portion of the right auricle and the coronary sinus. Eventually this primitive tube is converted into the His bundle, known to some as Gaskell's bridge, or the auriculoventricular bundle, which unites auricle with ventricle. Studies were first made on the hearts of amphibians and reptiles, where the beat originates from the sinus venosus; in mammals, however, according to Lewis, sinus and auricle are usually fused together.

In the lower vertebrates, the sinus venosus, auricular canal and

aortic bulb are still recognizable, but not so in the human species.

The remains of this sinus, however, have been discovered by Keith and Flack,[1] and located at the mouths of the venae cavae. Previously, Keith, His and others had found the primitive tube (which eventually becomes the bundle) extending over from auricle to ventricle. The node was first called from its discoverer, Tawara's node.[2] It is situated in the wall of the right ventricle, near

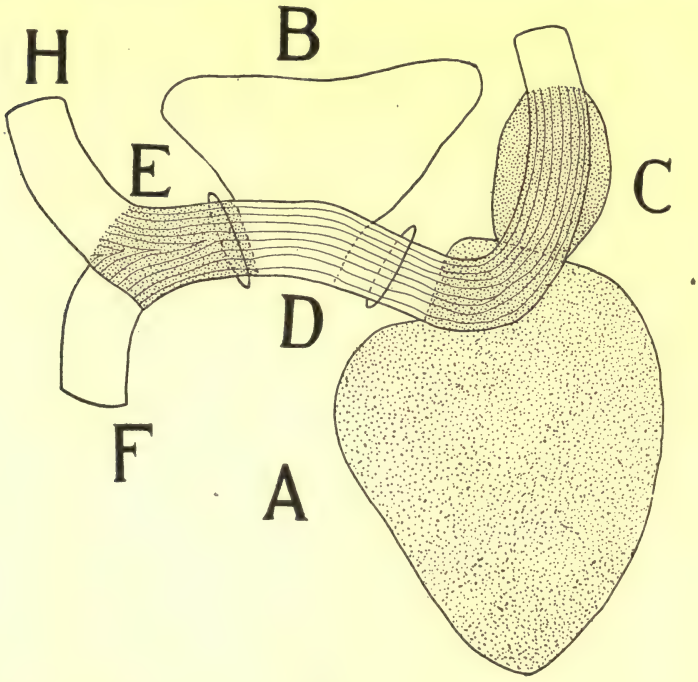

FIG. 1.

In Fig. 1 *D* is the primitive mammalian tube, indicated by longitudinal striations, extending from the sinus venosus *E*, where the upper vena cava *H* and the lower vena cava *F* join, through the bulbus cordis *C* to the aorta. *B* is the primitive auricle and *D* the auricular portion of the primitive tube, or auricular canal. The bulbus cordis is eventually included in the human right ventricle. (Schematic representation after Keith.)

the mouth of the coronary sinus. Tawara, Keith and Flack were able to trace the auriculoventricular bundle to the auriculoventricular septum, from which its branches extend into the walls of the ventricles.

[1] Keith and Flack (*Journal of Anatomy and Physiology*, 1907, XLI. 172-189.

[2] Tawara: Das Reizleitungssystem, Jena, 1908.

The bundle curves over to the membranous septum, entering and following the moderator band *F*, until it reaches the base of the large group of papillary muscles *G*. According to present views, this bundle of muscle-tissue connects auricle and ventricle functionally, the impulse originating in the auricle and passing gradually into the right and left groups of papillary muscles.

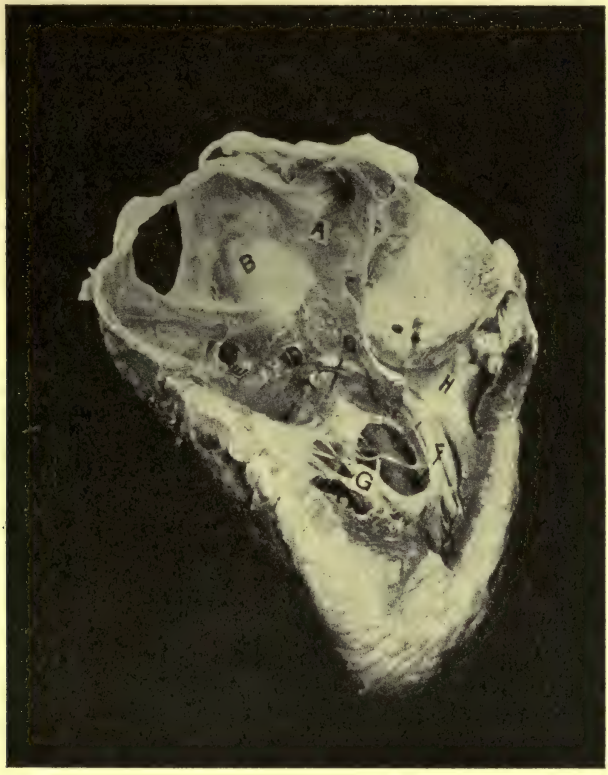

Fig. 2.

Human heart showing the origin, course and distribution of the auriculo-ventricular (His) system. The anterior walls of the right ventricle and right auricle have been removed. The intra-auricular septum, the tricuspid valve, the papillary muscles *G*, the moderator band *F*, and the interior of the infundibulum *H* are exposed. *A* lies in the right auricular appendix, *B* in the fossa ovalis, *E* is placed beneath the mouth of the coronary sinus. Directly beneath *D* is a fan-shaped bit of muscle; a bristle has been placed beneath it. From this point the auriculoventricular bundle and its right branch are traced as they lie on five bristles between *D* and *F*.

From a specimen in the possession of Keith.[3]

[3] Lewis: Mechanism of the Heart Beat, London, 1911.

Briefly, the work of Wooldridge, Tigerstedt, Gaskell, Kent, His, Retzer, Braeunig and Tawara[4] has proved that round about the coronary sinus and at the base of the septum there is a specialized group of auricular fibres now known as the auriculoventricular or Tawara's node. From this point the bundle runs at first almost horizontally forwards and to the left, ensheathed in a fibrous canal, and it pursues its course directly to the right of the central fibrous body of the heart, as far as to the membranous part of the septum of the ventricle. At the anterior part of this membrane the bundle divides, entering the left ventricle immediately beneath the centre of the aortic valve. Ultimately its branches are continuous with the subendocardial network of Purkinje's fibres which lines most of the interior of both ventricles.

The bundle thus constitutes the functional union between the auricle and ventricle, and it is through this structure that normally the impulse from the auricle originates and causes ventricular contraction. But the structure of the various divisions of this system varies considerably. At the auriculo-nodal junction the smooth muscle fibres are interspersed with connective-tissue, nerve fibres and ganglion cells. In their course the muscle fibres increase in size until they form networks, and finally take on the well-known character of Purkinje's fibres. Monrad-Krohn (*Norsk Mag. f. Laegevidenskaben.* LXXII. 1, Supp. 1-176) in dissecting the bundle of His in four cases of Adams-Stokes disease found fatty and vacuolar degeneration of the main bundle in one and deposits of lime compressing the muscle fibres in three.

Apropos of this point of origin for muscle contractions, at the meeting of the German Congress for Internal Medicine, in April, 1911,[5] Hering stated that he had found that the source of the contractions was not always at the auriculoventricular or sinus node, for after removal of the node in dogs and cats the impulse was not checked. In fact, he had found that the origin of the impulse might be in the auricle or ventricle, or some point between them, and constitute auricular, ventricular, or auriculoventricular extrasystoles. And he maintained that by the use of the electrocardiogram he could determine the precise auricle or ventricle in which the contraction took place. This was new. He also added that the point of origin of cardiac contractions might change. The

[4] For further references to their work, see Lewis on "The Mechanism of the Heart Beat," pp. 7-8.

[5] *Verhandl. des Deutsch. Congress.* 1911, XXVIII. 418-424.

result of accident to the node would be that some other area, whether in an auricle or a ventricle, would originate the impulse vicariously. That in other points than the auriculoventricular node contractions could originate was, however, previously known.

In extrasystole, according to Nicolai of Berlin, the point of origin is movable. For he holds that the normal electrocardiogram shows that the impulse moves from the base to the apex and then back again, in contrast with Hering's idea that it may originate in any of the four chambers or the septa. According to Nicolai[6] there are three types of extrasystole: (1) That which originates

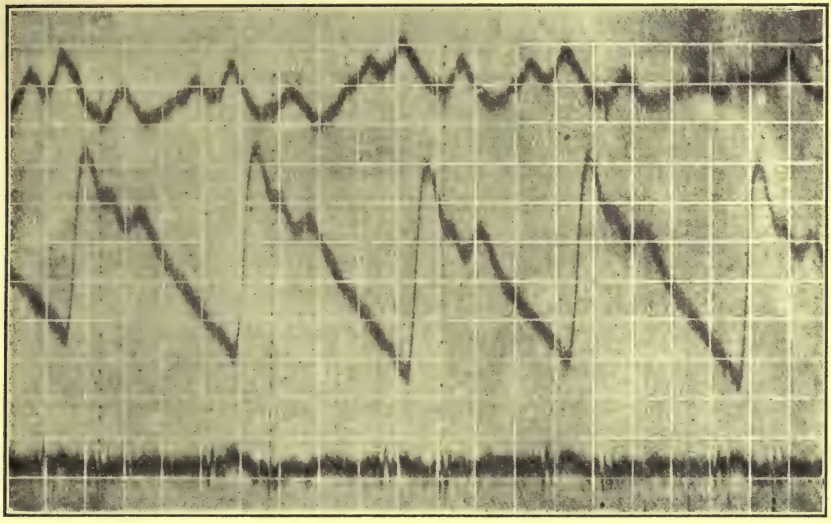

Fig. 3.

In Fig. 3 the upper tracing represents the jugular pulse, the middle the radial, the lower the heart sounds as photographed. By following with the eye the vertical white lines, the relations in time between the heart-sounds and the radial and jugular waves are seen.

about the apex, (2) that originating about the base, and (3) that where the point of origin is in the right ventricle.

The Einthoven type of electrocardiogram, he claims, is derived from the left heart exclusively. In fact, by means of physiological experiments he has undertaken to show that the wave starting from the left heart near the auricle gives the classical Einthoven curve, while the wave starting from the apex in the right ventricle is of the

[6] *Verhandl. des Deutsch. Congress,* 1911, XXVIII. 221-226.

inverse type, which instead of the ascending limb has the descending limb or the so-called reverse type of electrocardiogram.

One of the most interesting of recent developments has been that of Ohm of the Kaiser Wilhelm Institute of Berlin.[7] Working under Kraus, he has been able by special apparatus to photograph the heart-sounds on paper, in the form of waves, contemporaneous with tracings of the arterial and venous pulses. This method might be called phonophotography. The preceding illustration, Fig. 3, shows the three tracings registered simultaneously by him.

The first heart sound is seen to be contemporaneous with the beginning of the systolic radial wave and the highest wave of the jugular. The second heart-sound corresponds in time with the third wave of the jugular and the dicrotic notch of the radial tracing. The first wave in the jugular tracing, on the other hand, corresponds to the presystolic wave of the radial; and Ohm would call it therefore the presystolic wave. The second or highest wave of the jugular, owing to its relation in time with the systolic upstroke of the radial, he would call the systolic wave; while the third wave corresponding in time with the dicrotic notch, he would call the dicrotic wave.

There is a minute difference in time between the first heart-sound and the second or highest jugular wave; the third wave of the jugular actually precedes the second heart-sound by 1-24 second.

But, in taking the carotid and jugular pulses conjointly, he has found an exact correspondence in time between the diastolic wave of the jugular with the dicrotic wave of the carotid and the second heart-sound. This dicrotic point, he maintains, corresponds to the closure of the aortic valves.

Auricular fibrillation, as a cause of irregularity of the pulse, has been recently described by James Mackenzie (Hare's "Modern Treatment," 1910, I. 22), where he says he is now prepared to call by the name auricular fibrillation what he previously named nodal rhythm—the permanently irregular pulse of Hering. He further defines this condition as one in which the cardiac cycles vary continually in length, there being no sequence of beats having the same length. Another criterion is that, on the appearance of the irregularity, the auricular systole stops. Mackenzie holds that auricular fibrillation exists in 70 to 80 per cent. of arrhythmias. In 1902 he ascribed this condition to paralysis of the auricle, but having found that there was actually a simultaneous contraction of aur-

[7] *Verhandl. des Deutsch. Congress*, 1911, XXVIII. 331-333.

icles and ventricles, he put the source of the difficulty at the auriculoventricular node, which governs auricular and ventricular contractions through the bundle of His.

In 1905 Cushing and Edwards suggested that in some of these patients the cause might be auricular fibrillation, a condition in which component parts of the muscle wall of the auricle contract independently of one another, and in such a disorderly fashion that it might almost be said auricular contraction as a whole was at a standstill. But it was not until 1909 that researches by Lewis on the lower animals showed, by means of comparison between the arterial and venous pulse-tracings and electrocardiograms, that this so-called nodal rhythm, or permanently irregular pulse, was to be attributed to auricular fibrillation.

Now in fibrillation there seems to arise in the auricle a continuous shower of stimuli, which falling on the node excite it to send stimuli to the ventricle as rapidly as the bundle (and so the ventricle) is capable of taking them up. At first the ventricular contraction is apt to be very rapid, and the patient may soon die of heart failure. But if the ventricle can be made to beat more slowly, the patient may lead a useful and even vigorous life for some years. It is therefore very important to diminish the rate, and this is done by digitalis in a remarkable manner. The gravest sign is an increase in rate; say from 100 to 150. The digitalis should then be pushed until there is a fall to 80. My experience tallies with Lewis's view; for, in the permanently irregular pulse, relief only comes from the continuous use of digitalis or strophanthus. Hering recognizes this fact. Mackenzie believes that a good deal can be done for the patient. He finds, for example, that the irregularity is most often associated with rheumatic hearts, usually those of mitral stenosis, and with the fibroid heart of senility. In fact, as special deposits are common in both these varieties of cardiac disease, it seems possible that the deposits are the cause. In one case of mine, there was a fibroid tumor of the uterus; its removal did not improve the cardiac difficulty, however.

According to Lewis, auricular fibrillation constitutes 50 per cent. of all irregularities, the disturbance of cardiac rhythm having its origin in the auricle, and being due to temporary or permanent incoördination of the musculature of this chamber.

In a study of 106 cases, he has reached the following conclusions, which vary, however, somewhat from those of Mackenzie. The character of the radial pulse is very striking. The rate may

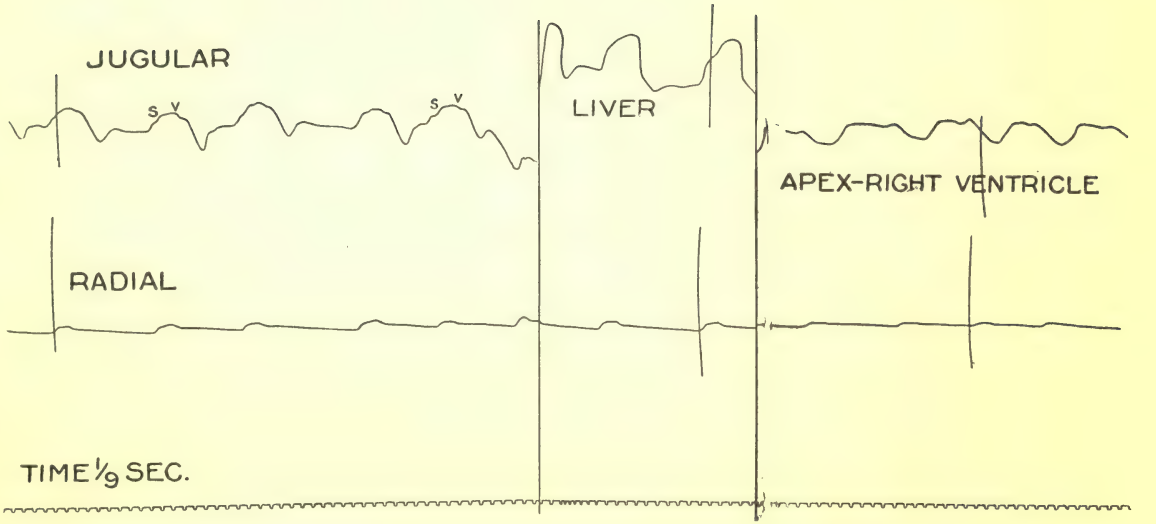

FIG. 4.

In Fig. 4[8] is seen the jugular tracing of what is now called auricular fibrillation, formerly called by Mackenzie nodal rhythm in a patient with a double mitral lesion, tricuspid regurgitation and auricular paralysis. The apex beats are irregular, while the auricular waves are absent in this particular tracing.

S is Mackenzie's auricular wave *A*, and *V* is Mackenzie's carotid wave *C*.

[8] Newer Conceptions of Cardiac Arrhythmias (*Medical Record*, May 15th, 1909).

be reduced as low as thirty, or increased to as high as 200, but the rate in itself has little significance, because many beats of the heart may not reach the radial artery. But the fast rates, viz., between 110 and 150, are the most common, and with these the irregularity is greatest. Now, according to him, there are two criteria of auricular fibrillation: (1) Seldom or never in any one case do cycles of the same length succeed one another; and (2) there is no definite or continued relationship between the length of a beat and the pause which precedes it. These two criteria are shown in the tracings of Fig. 4.

Lewis holds that normal auricular action may be in abeyance, while the peripheral circulation is unhindered. He holds that in the vast majority of instances, a sphygmogram showing that no two successive heart-beats are of the same length means the diagnosis of auricular fibrillation (Fig. 4). To Cushny, Mackenzie, Wenckebach, Rothberger, Winterberg and Lewis, the credit of the discovery of auricular fibrillation is due[9]. Janowski[10] has known such a case to last five and a half years, Mackenzie ten years.

The central nervous system is known to furnish the heart with two sorts of nerve fibres. One of these, known as the inhibitory, reaches the heart through the medium of the vagus. Their activity will slow or even arrest the heart's action. Luciani calls the vagus the diastolic nerve of the heart, its stimulation causing dilatation of both auricle and ventricle.[11] The second sort of fibres reaches the heart through the medium of the sympathetic system. They are known as accelerator fibres because they quicken the heart's action. The upper and lower branches of the vagus unite in the heart with the sympathetic network so as to form the cardiac plexus, filaments from the vagus, however, terminating in the sino-auricular node, i. e., the remains of the sinus venosus.

Both vagus and sympathetic fibres are efferent in character, but there are also afferent filaments, carrying sensations away from the heart. Some of these are stimulated at each beat of the

[9] Cushny (*Amer. Journ. Med. Sciences*, 1907, CXXXIII. 66-77).
Mackenzie: Diseases of the Heart, London, 1908.
Wenckebach (*Archiv. f. Anat. und Phys.*, 1907, Phys. Abth. 1-24.
Rothberger and Winterberg (*Wien. klin. Wochenschr.*, 1909, XXII. 839-844.
Lewis (*British Med. Journ.* 1909, II. 1528).

[10] Janowski: Functionelle Herzdiagnostik, Berlin, 1910.

[11] Luciani: Physiologie des Menschens, Muenchen, 1905, I. 168-173

heart. These latter fibres may cause painful sensations, for the stimulated vagus may send radiations to various sensory nerves. Pain in the gums and throat may be due to radiations from the vagus to the fifth nerve. But the cardiac plexus lies on the arch and ascending portions of the aorta, and from it the heart receives both its inhibitory and accelerator fibres.

Now, as the result of a very large number of experiments, it seems certain that the vagus affects both the rate and the force of cardiac contractions, and also the conductivity of contractions that normally pass from the auricle to the ventricle. The familiar experiment of pressure on the vagus in the neck causes, it will be remembered, slower and stronger cardiac contractions, while, on the other hand, if the auricle is injured in any way or pressed upon, permanent or temporary loss of conductivity, as the case may be, is liable to follow. Indeed, experiments have shown that injury to the auricle or pressure on it may so disturb the conductivity that, of several auricular contractions, few or perhaps only one ventricular contraction may follow; in other words, there will be heart-block. Direct stimulation of the vagus in the lower animals may even keep the heart inhibited for some time[12]; or the inhibition may be indirect, and due to a blow on the abdomen, or distention of the stomach or intestines by gas. This latter accident can cause the heart to stop entirely. I have seen a case of complete temporary heart failure of this kind, in which the respiration also was totally suspended synchronously.

Interesting accounts of resuscitation have appeared in the daily papers, where in one instance an asphyxiated miner was restored to life, after both cardiac and respiratory action had been suspended for some hours, by the removal of blood and the substitution of normal salt solution, together with the use of a machine called the "pulmotor," which first removes deteriorated air from the lungs and then pumps in normal air. I have as yet seen no scientific description of the method, however.

A probable explanation of the phenomenon of inhibition by Howell[13] is that the afferent impulse conveyed to the central nervous system stimulates those nerve cells in the medulla that give origin to the inhibitory fibres causing cessation of cardiac action; and yet, too, an afferent impulse may excite the activity of the accelerator nerves, by reacting on their roots, presumably somewhere in the

[12] Mills (*The Nervous System, etc.* 1898, 117.)
[13] Textbook of Physiology, Phila., 1911.

brain. The group of cells from which the vagus arises comprises the so-called cardio-inhibitory centre, but its anatomical site is unknown. These cells in health should presumably be in constant tonic activity; and it is through their influence that the rate of the pulse is kept down by their opposition to the activity of the accelerators. For the rate of a pulse is due to the resultant of the antagonistic forces of these two opposing agencies. Furthermore, recent discoveries by Hemmeter, to be alluded to later, show that there are both inhibitory and accelerator ganglia in the heart itself. The advantage of such a "balanced mechanism" is that each of the antagonistic forces is peculiarly susceptible to a stimulus.

Though we are obliged to theorize from a rather limited amount of knowledge as to the anatomical site of these nerve impulses, this much we know: that the rate of cardiac contraction depends on various conditions, such as sex, age, size, blood-pressure, muscular exercise, and the composition of the blood. For we know that women have a higher rate than men, large individuals have a slower rate than small ones, and, while the average rate at birth is set at 140, it falls gradually toward 70 in senility, rising again gradually towards 80 in extreme old age. Also, as blood-pressure in the arteries falls, the pulse-rate rises, while as the pressure increases the rate falls. So, too, the rate increases under vigorous muscular exercise. In some alterations of the blood, as in asphyxia, where the CO_2 content rises, the pulse-rate first increases and then decreases, as soon as toxic symptoms supervene (Howell). These facts should always be borne in mind in estimating the significance of a high or low pulse-rate.

Bearing on this matter, the following interesting facts have been reported to the writer in a private letter relative to work done at the Marine Biological Laboratory at Wood's Hole, Mass., during the summer of 1911.[14] Experiments on sharks, dogfish, and sea turtles to the number of 346 demonstrated the existence of both accelerator and inhibitory ganglia in the sinus venosus and auricles of their hearts. Stimulating the inhibitory ganglion from a normal of 36 would slow or arrest the heart, according to the intensity of the electric current. Section of the vagus, however, caused no acceleration in the sharks and dogfish; therefore, the vagus was not in tonic activity.

[14] Prof. J. C. Hemmeter, of the Department of Physiology at the University of Maryland.

It was incidentally found that iodothyrin favors vagus inhibition. This suggests a remedy for the pulse of persistent arrhythmia, where a reduction of the rate is essential in the treatment. It was ascertained, further, that inhibition of the heart by the vagus is not due to a chemical substance, such as potassium chloride, in these animals, at least; for the experimenter connected the aorta of one shark with the auricle of a second shark, so that the blood from heart No. 1 went into heart No. 2, and then stimulated the vagus of No. 1, which was brought to one-half its former rate and finally stopped, but without producing any change of rate whatever in heart No. 2. If vagus inhibition had been due to any chemical substance, it should have passed into heart No. 2, whose vagus was not stimulated, and slowed it, but it did not.

From the above recital of some of the more important discoveries that have recently been made in matters relating to the anatomy and physiology of the heart, it is apparent that each has an important bearing on diagnosis and treatment, while it is also equally plain that there remain fruitful fields for further research along the same general lines.

Ventricular fibrillation, or flutter, is a subject even less understood by physiologists than the same condition occurring in the auricle, but a few facts with regard to it have recently been established. This has been accomplished by means of experiments upon the lower animals, observations having been made conjointly by the electrocardiograph and the myograph. Lewis (Mechanism of the Heart Beat, London, 1911), experimenting on dogs, discovered that although neither irritation nor section of the vagus resulted in fibrillation of the ventricle, he could bring about premature ventricular contractions by either tying the descending branch of the left coronary artery or obstructing the flow of blood in the right coronary. These premature contractions occurred at increasingly briefer intervals, at first one at a time, then in groups. As the irregularity increased, the pulse rate rose rapidly, increasing to 300 and at times to 420, actual delirium cordis finally appearing in some cases. Lewis also found that he could produce the same result by irritation of either a large papillary muscle, the lower angle of the right ventricle, or the central surface of the latter. The normal pulse rhythm, although overcome for the time by the extraneous impulses (known as heterogenetic or ectopic), usually reappeared when the irritation or obstruction producing the abnormal condition was relieved.

Further, Lewis and Levy (Heart, Vol. III, 1911-12), experimenting with chloroform anesthesia in cats, found that ventricular fibrillation occurred, followed by heart failure. It could also be produced in the same animals under light chloroform anesthesia, by intravenous injection of a one-quarter to one minim dose of adrenalin chloride in a 1 : 1000 solution, although similar experiments under full anesthesia produced no such results. As the subjects in whom the administration of adrenalin produced ventricular fibrillation seldom entirely recovered, a practical deduction from these experiments would seem to be that intravenous injections in a patient under light chloroform anesthesia are attended with great risks. We may regard it therefore as demonstrated that ventricular fibrillation may result from obstruction to the circulation in the coronaries, or from any injury to the substance of a ventricle.

Ventricular fibrillation has been once demonstrated in a human subject with paroxysmal tachycardia by Hoffmann (Heart, Vol. III, 1911-12), by means of the electrocardiograph. As the patient recovered, it is evident that the condition is not necessarily fatal.

Chapter II.

SPHYGMOMANOMETRY AND SPHYGMOMANOMETERS.

In the differential diagnosis of cardiac diseases, the thermometer, the microscope, laboratory tests, and the various instruments for determining blood pressure and for registering cardiovascular action give us valuable assistance. But there is a wide difference in their comparative usefulness. While the clinical thermometer is accurate and gives results of absolute value, the others yield information that is less positive.

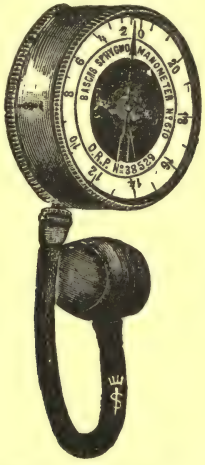

Fig. 5—Von Basch's Instrument.[1]

A sphygmomanometer demonstrates blood pressure. The instrument was probably first devised by Harrison, in 1834. Improvements by Vierordt, Kries and Marey followed, but the first clinical model seems to have been that of von Basch.

Figure 5 shows the general plan of his instrument. This model was made public in 1887. (*Berl. Klin. Woch.*, 1887, XX. V. 181). It consists of a metal cylinder, connected by means of a bit of rubber tubing terminating in a hollow ball or pelote, with the dial, through the medium of a spiral spring. Von Basch at

[1] Kny-Scheerer Co., 404 W. 27th St., New York City.

first used water to actuate the spiral spring, but later air. By means of aerial pressure he made the membrane bulge a little, thus transmitting the oscillations of the pulse waves to the manometer with more delicacy than when water was used.

The method of procedure was as follows: The bulging end of the pelote was placed over the artery, the temporal or radial, as the case might be. The index finger, pressed on the vessel, adjacent to the pelote, but on its distal side, gradually obliterated the pulse. The amount of pressure registered by the manometer as sufficient to stop the pulse was taken to be the *maximum,* or so-called *systolic* pressure, this criterion having been given by Marey. It has been generally adopted.

Von Basch found, however, that the maximum pressure of the temporal artery taken by his instrument averaged between 90 and 120, while that of the radial varied from 110 to 160—tolerably wide differences. According to present methods, the actual maximal radial pressure in normal children varies somewhere between 95 and 110; in normal adult females between 115 and 125; in normal adult males between 125 and 135.

Potain made improvements in von Basch's instrument. He reinforced the rubber ball with extra layers of rubber cloth, in order to give the ball greater firmness.

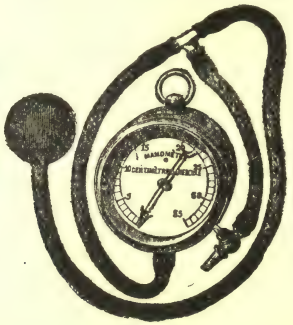

Fig. 6—Potain's Instrument.[2]

In Figure 6 is seen the rubber ball or pelote attached by means of a firm rubber tube, fitted with a stop-cock to a metallic manometer, which has been corrected by a U-shaped mercurial instrument, registering in cubic centimeters of mercury. The

[2] Kny-Scheerer Co., N. Y.

rubber ball is pressed against the radial, and it in turn against the bone. This model was first described in the *Archives de Physiologie,* 1889, 555-569.

Potain maintained that with his instrument the maximal pressure was not so important to determine as the mean pressure; a point that is true and that will be alluded to later. But he still failed to secure satisfactory records, as may be seen by the fact that in a young girl whose temporal pressure was recorded as 5.5 centimeters the radial was recorded as 16.5. In another individual (an adult) the instrument registered 12.5 for the temporal and 19.5 for the radial.

As early as 1888, Marey attempted to overcome this inaccuracy. By his plan he compressed the vessels on all sides instead of on one side as heretofore. Constructing a tight metal box and filling it with water, he introduced the hand and forearm, which closed the opening. This water box was connected with a mercurial manometer and a recording drum, and also with a reservoir, by means of which the water pressure could be increased at will. The oscillations in the mercurial column actuated by the arterial pressure increased in amplitude up to a certain point, after which they declined. Before they entirely ceased the hand blanched, showing that the vessels had collapsed and that the systolic pressure was overcome. Marey reasoned that at the moment when the oscillations reached their maximum the external pressure and that within the vessels were exactly equal, and that their walls, being relieved of any tension on any side, would perform their maximum oscillations. This criterion of Marey has been accepted as indicating *standard maximal pressure.*

Marey used Fick's method, described by the latter in 1869. (Marey, La Circulation du Sang, Paris, 1881, 199.) Fick's was a modification of the apparatus of Chelius. (*Vierteljahresschrift f. Prakt, Heilkunde* 1850, II. 92.)

The recording drum was the invention of Ludwig, and is called Ludwig's Kymographion. The apparatus, as used at that time, consisted of a U-shaped manometer, having on the surface of the mercurial column a float connected with an ivory rod, attached to which, at right angles, was a recording pencil, which inscribed the oscillations of the mercury on a revolving drum or tambour, covered with smoked paper. The drum was actuated by a weight. Ludwig was the inventor, apparently, of the graphic method. By means of this method as applied to the smoked paper

on the drum, the degree of systolic and diastolic pressure is shown with accuracy.

Mosso, wishing to improve on Marey's plan, devised an instrument which he described in 1895. (*Arch. Ital. de Biol.*, 1895, XXIII., 177.) He aimed, as Marey did, not to measure the external pressure necessary to obliterate the pulse, but the external pressure under which the arteries developed their maximum amplitude. His instrument consisted of the usual U-shaped manometer, a water reservoir, and two metal tubes, into which the middle and ring fingers of the hand were introduced. The metal tubes were then filled with water, and to prevent its escape the fingers were inserted into loose rubber glove-fingers attached to the tubes. The two metal tubes were connected not

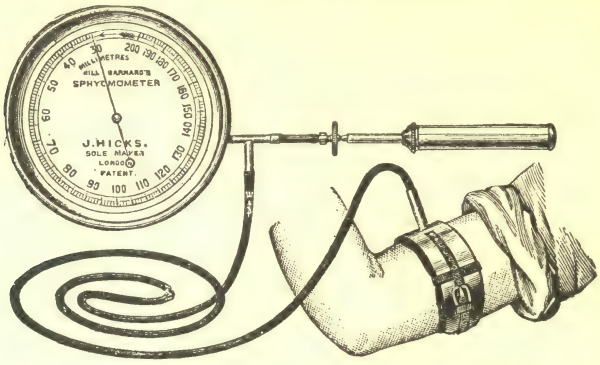

FIG. 7[8]—The Hill and Barnard Sphymometer.

only with the reservoir, but also with each other by a smaller tube, and with a pump by means of other tubes. By means of a crank acting through a piston, water was pumped into the tubes, the air meanwhile escaping at the vent. The piston was compressed until the oscillations of the mercurial column indicated maximal (systolic) pressure. The record was then read off. Compression of the fingers was subsequently utilized by Gaertner in his apparatus, while the pump was used in that of Muenzer. (*Zeitsch. der Exper. Path.*, 1907, IV., 1, 138.) Mosso's apparatus was, however, unsuited for clinical work, and was too complicated for general use.

The Hill and Barnard sphygmometer is the simplest of all. (Fig. 7.) It consists of an arm-band, connected by rubber tub-

[8] Kny-Scheerer Co., N. Y.

ing with a metallic manometer, the air being pumped into the tubes by an arrangement like a bicycle pump. This instrument measures in millimeters. The armlet, however, is too narrow and the tubing too light in weight.

In 1899 Gaertner devised his tonometer (so-called from *tonos,* vibration). The finger of one hand is introduced into a hollow rubber ring connected with a mercurial manometer; the ring is passed over the first joint and the tissues of the inguinal phalanx are compressed by smaller rings passed over it, until the skin of the phalanx is blanched. The hollow ring is then inflated, by means of a Politzer bag, until the blood vessels are empty, after which the small rubber rings are slipped off. The reading of the manometer is noted, and the air is gradually admitted until the blood begins to return to the blanched capillaries of the fingers. The point at which the blood begins to return is held to be the *maximal* or *systolic* pressure.

There have been many subsequent models of this instrument, of which I have used several at different times. As the early models only recorded maximal pressure, Sahli (*Lehr. der Klin. Untersuchungs Methoden,* 1903) modified his method by the use of the U-shaped mercurial manometer which registers minimal (diastolic) pressure. He made the long uprights of two pieces for convenience in carrying. In hospital and office work, however, the long tube should be made in one piece; the jointed tube is apt to accumulate dust, and is more easily broken.

The difficulty with the Gaertner method is that the finger should be warm, the ring fit tightly, and there should be several rings so as to be adapted to fingers of various sizes; while pressure may produce paralysis of the blood vessels, so that the blood may be slow in returning. Compression by the arm-piece is more satisfactory. In fact. it may be said that in newer instruments the finger rings have been rejected.

The Riva Rocci instrument has been described as follows:

"A closed system of air connects a rubber bulb held by the operator with a rubber band placed around the arm or leg of the patient and a mercury manometer. By the law of gases, equal pressure is transmitted to every point throughout the air system. When the pressure is raised by the operator to such a point that the pulse of the patient distal to the constricting band is obliterated, the height of the mercury column in the manometer is equivalent to the maximum arterial blood pressure.

"Any one at all trained in pulse palpation can make an accurate reading at the first trial. An estimation takes from fifteen to thirty seconds. The arm-piece is placed around the patient's upper arm, midway between elbow and shoulder, and adjusted to fit. The operator, with one hand, increases the pressure by squeezing the rubber bulb, and, with the other hand, palpates the patient's radial at the wrist. When the pressure just obliterates the pulse at the wrist, the height of the mercury is noted,

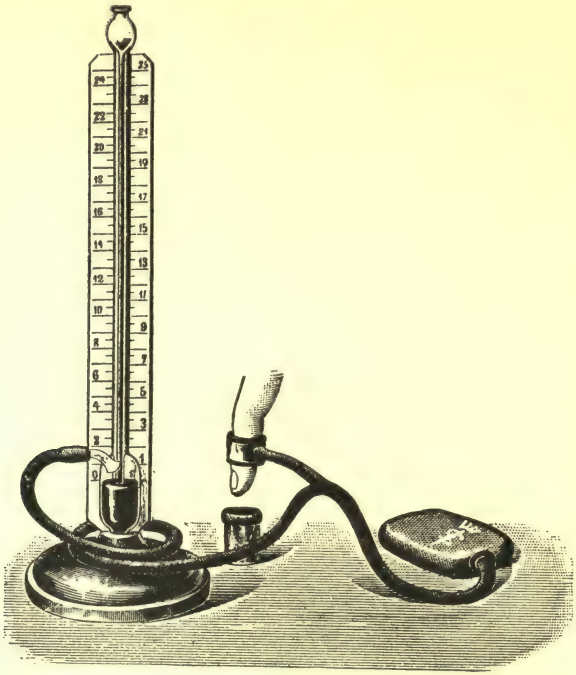

Fig. 8—Gaertner Tonometer.[4]

and it is then allowed to drop slowly until the pulse returns. This maneuver is repeated without letting the air out, and by merely squeezing and releasing the bulb. The point above which the pulse is obliterated and below which it returns is the reading of maximum arterial blood pressure. A determination within two or three millimeters should be considered satisfactory.

"A reading of mean arterial blood pressure may be made with this instrument, as described by Professor Gumprecht, by finding

[4] Kny-Scheerer Co.

the point when the greatest excursion of the mercury column occurs during cardiac systole after clamping off the tube leading to the reservoir bulb.

"The normal maximum blood pressure averages, when lying at rest:

For children of 1 to 3 years, 85 to 95 mm.
For children over 3 years, 95 to 110 mm.
For adult females 115 to 125 mm.
For adult males, 125 to 135 mm."

Lauder-Brunton (*Lancet* 1908, II. 1126-1133) has put the maxi-

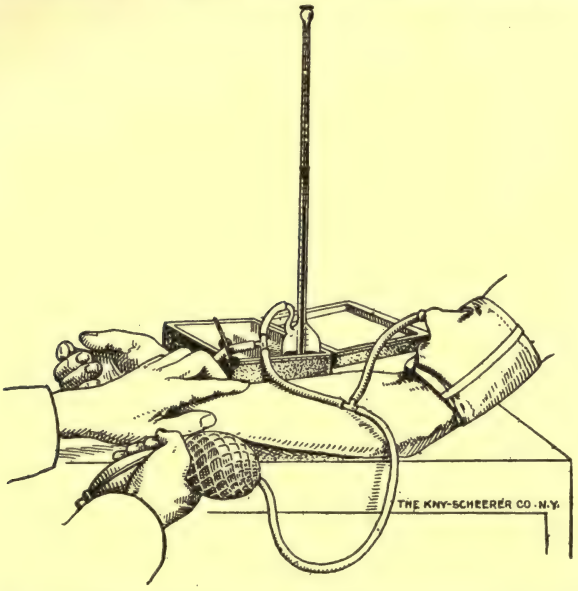

Fig. 9—Cook's Modification of the Riva Rocci Instrument.

mum pressure in children from 8 to 14 years of age at 90 millimeters of mercury; from 15 to 21 years of age at 100-120; from 21 to 65 years from 120 to 135 or even 150. In women he has put the pressure as a rule at 10 to 15 millimeters lower than men.

Gibson (*Edinburgh Med. Journal,* 1911, N. S. VI. 197) puts the normal systolic pressure in the young male adult at from 90 to 130 mm., while the diastolic is from 70 to 100. These figures are less in women and still less in children. But recorded pressures vary somewhat with the kind of instrument used, as we have seen, as well as with age and abnormal conditions of the system.

Cook's modification of this instrument is shown in Figure 9.

It consists of an upright manometer mercurial tube, terminating in a bulb, which when in use rests in a wooden socket or stand. To the bulb is attached rubber tubing, connected with two rubber bulbs. This tubing is also connected with a rubber ring about two inches wide, which encircles the arm. The instrument should preferably be used when the patient is in the recumbent position. There are three objections to this model: the arm band is too narrow for suitable compression, and the tube is delicate and lia-

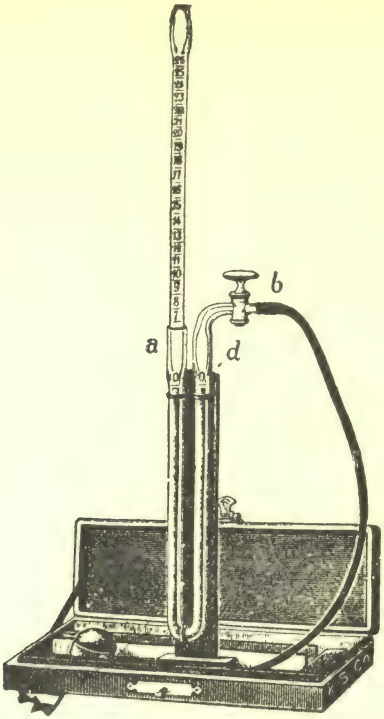

Fig. 10—Sahli's Instrument.[5]

ble to be broken. The bulb has to be filled with mercury from time to time.

The first two of these objections are overcome in the Sahli (Figure 10) and Stanton instruments. The latter also has advantages over the Riva Rocci model, in that it is more durable, easily and quickly managed, and more portable. It is not necessary to remove the mercury from the tube.

[5] Kny-Scheerer Co., N. Y.

Stanton's armlet is made of canvas, reinforced with strips of tin, and held in place by two leather straps. This armlet is connected by means of a stiff rubber tube with a metal manometer, to which the column of mercury is fastened as in an ordinary house thermometer. A cistern communicates by means of a metal tube with the mercurial column. The screw cap of the cistern (B. Stanton, *Univ. of Penn. Bull.* 1903, XV. 466) is provided with a metal valve and is connected also with the rubber armlet at one end and at the other with a rubber bulb used as an air-pump. A stop-cock shuts off the air in the rubber tube, while a screw valve allows the air to escape slowly from the closed air system, and the mercury to return to the cistern.

The air is pumped in by a double bulb syringe similar to that used with the thermo-cautery. A variation of 5 to 15 mm. of mercury may be expected in cases of high pressure; otherwise the instrument has a high degree of accuracy.

This instrument has been extensively used in this country. It is made by the Arthur H. Thomas Co., 12th and Walnut Sts., Philadelphia

Fellner, an assistant of the late Nothnagel, undertook a few years ago to compare the readings of the Riva Rocca instrument, as modified by Strasburger and used on the human species, with absolute results obtained in laboratories by experiments on the lower animals.

They were conducted contemporaneously on the same animal with two instruments, one canula being introduced into one vessel and another into another. Five animals were used. A Huerthle tonograph was used for one vessel and a Riva Rocci for the other. The differences were alarming. On the other hand the *pulse pressure,* i.e., the difference between the maximal and minimal, was very small, on an average 3 mm.

The *pulse pressure,* he held, therefore (*Deutsch. Arch. f. Klin. Med.* 1906-7, LXXXVIII. 1-35) represents the true size of the pulse.

The best way to get an idea of the value of the instrument is to test it on oneself. From my experience, the Riva Rocci maximal systolic pressure varies from 30 to 40 mm. of mercury in a single day, and the diastolic still more—40-45. Indeed, there may be a variation of 40 mm. in 10 successive trials. But there is no doubt that the variation is less in the newer instruments. The

von Recklinghausen (Figure 11) is an example of a superior instrument, but it is not fitted for clinical use.

The sphygmomanometer is particularly useful in determining the effects of drugs, as was shown by the studies of H. W. Cook. (*Journal of the American Medical Association,* 1908, L. 676-679.)

A substantial and at the same time portable instrument is that of Janeway. It has a fairly wide armlet such as was introduced by von Recklinghausen, the U-shaped tube of Ludwig, of which the long arm is jointed after the manner of Cook's modi-

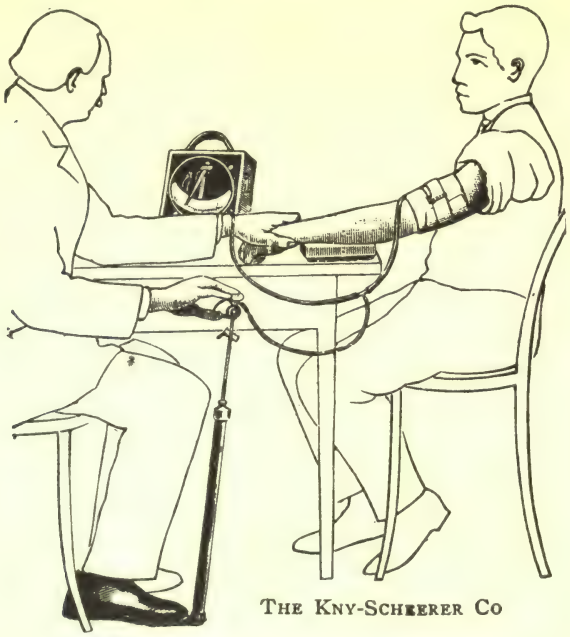

FIG. 11—Von Recklinghausen's Instrument.

fication of the Riva Rocci, while the Politzer bag is substituted for the double bulb of the Riva Rocci. By inserting a rubber cork into the ascending arm of the mercurial column, after the upper section has been removed, the mercury is kept in place.

This instrument records both systolic and diastolic pressure, is simple, durable, and easily manipulated. It is said to record with an error of not more than 6 to 10 mm. of mercury. A point of importance is the stop-cock, which materially aids in controlling the pressure. By turning it the air may be allowed to escape slowly through the needle valve, thus regulating tension in the

otherwise closed air current. The following rule is given for finding the diastolic pressure:

After obtaining the systolic allow the pressure to fall 5 mm. at a time, through the needle valve, watching the pulsation of the mercury column at each point. This will increase for a time, then

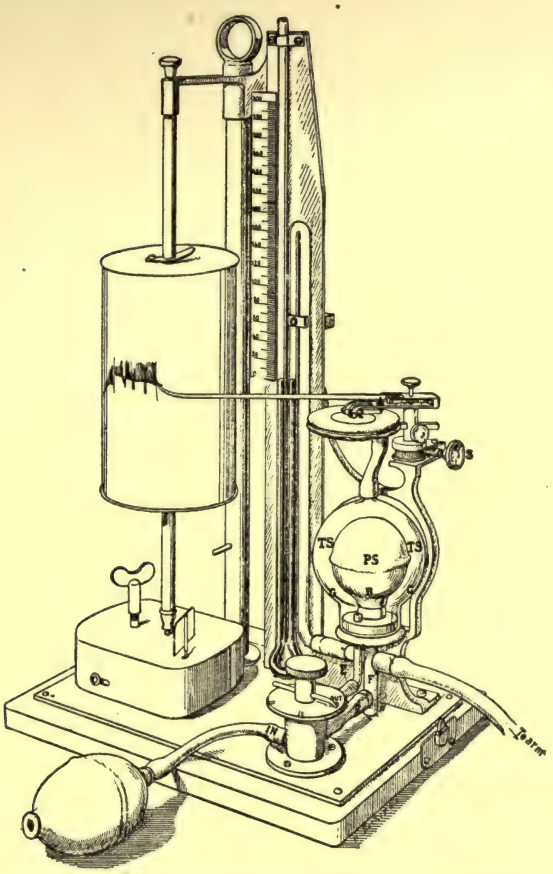

Fig. 12—The Erlanger Sphygmomanometer.

continue of the same extent for some millimeters. At a definite point the height of pulsation will decrease rapidly. The last point at which it is greatest is the diastolic pressure (criterion of Marey). Two or more measurements of this should be made.

One of the more recent models is the Erlanger, Figure 12. (*Johns Hopkins Hospital Reports,* 1904, XII. 62.) It makes use of

the armlet, the Politzer inflater, and the U-shaped manometer, but has in addition a Ludwig kymographion and tambour, the interior of which is in direct aerial connection with the tubes and chambers which convey the air to the mercurial column. By the use of the kymographion there is an accurate record of both systolic and diastolic pressure. The apparatus is elaborate and better suited for the laboratory or hospital than for private use.

It may be interesting to note some of the more important conclusions that were reached by Erlanger. When an individual after lying down assumes the sitting position, the pulse rate increases, but the *pulse pressure* diminishes. Eating at meals in-

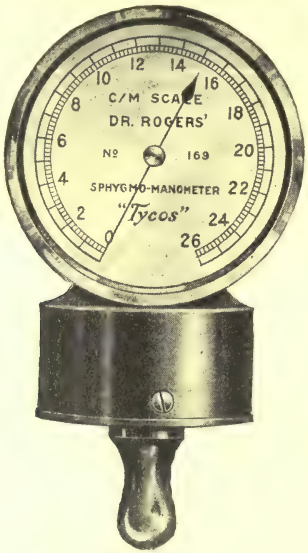

Fig. 13—The "Tycos."

creases pulse pressure and pulse rate. Pulse pressure increases throughout the day, but is small in the early part of the day.

Von Recklinghausen introduced his improved instrument in 1901. (*Arch. f. Exper. Path. und Phar.* 1901, XLVI. 78-132.) His broad band for the armlet has been adopted in principle for the more recent instruments The band should have a width of at least 5-6 inches, and be made of stiff canvas rather than flexible canvas or leather, as neither of the two latter grasp the arm as well as the von Recklinghausen. It fits closer and more comfortably, and without a close and firm apposition the reading of the column is marred. In

place of the Politzer bag introduced by Erlanger, von Recklinghausen used a hand pump. This idea came to a further development in 1906 (*Arch. f. Exper. Path. und Pharm.*, 1906, LV. 375-504) when a delicate bicycle pump moved by the foot replaced the hand pump, and a metallic manometer was used. This sort of apparatus is not clumsy, and can be carried in the hand. Air pressure is used. It is more sensitive than mercury.

There is an aneroid instrument in extensive use that is an improvement upon the Potain,—the Tycos of Rogers. This answers well for comparative measurements of pressure, is cheap, may be carried in the pocket, and is used extensively by the medical examiners of insurance companies. (Figure 13.)

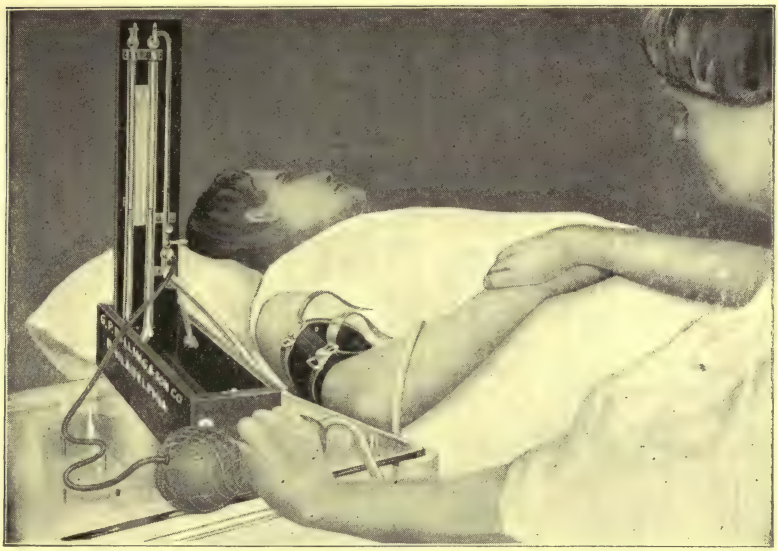

Fig. 14—The Faught Instrument.[6]

It consists of a gauge, a sleeve, an inflating bulb, and two short pieces of rubber tubing. These, when assembled and ready for use, constitute an instrument which is simple and so compact as to be contained in a leather case about the size of a physician's pocket case. It is said by the maker that for accuracy and sensitiveness the "Tycos" has all the advantages of the best mercurial instrument; that for compactness and durability it far surpasses them; and that it is practically indestructible.

[6] Made by the George P. Pilling & Son Company, of Philadelphia, Pa.

The model of the Faught instrument, shown in Figure 14, is simple, strong, and not likely to get out of order. It employs a U-shaped mercurial column, and is not susceptible to uncontrollable variation caused by atmospheric changes and other causes. It is deservedly popular.

My own modification of the Riva Rocci instrument is shown in Figure 15.

It has the long, unjointed Riva Rocci tube, but it is attached to the door of the box, so that when open, the box and door make a firm support for the tube. In order that the mercury may re-

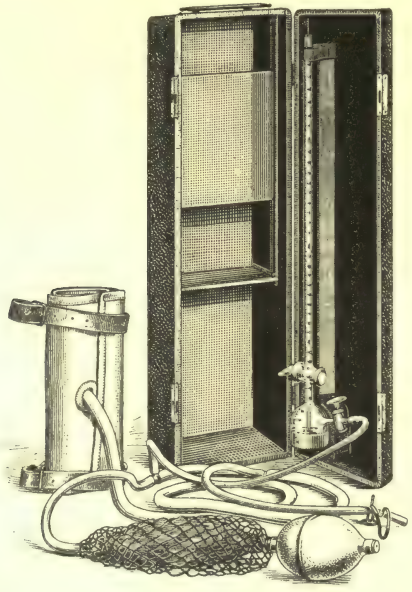

Fig. 15—Author's Office Sphygmomanometer.[1]

main in the reservoir when not in use, the latter is fitted with two glass stopcocks. Violent oscillations in the mercurial column are prevented by using a rubber tube that is unusually heavy. A broad cuff, 7¼ inches in width, something on the pattern of the von Recklinghausen, but larger, is used. Connected with the heavy tubing is a short, light, flexible rubber tube fitted with a clamp to regulate the ingress and egress of air. Air is pumped into the cuff by the double bulb of the thermo-cautery apparatus.

[1] Made by E. Machlett & Son, 143 E. 23d St., New York City.

When the box is opened, and the cuff and tubings are removed, they are all connected and available for immediate use.

In my instrument the question of diastolic pressure is of little account. Under the compression of the rubber bulb the column of mercury rises slowly as the pulse fades away, and when it has disappeared the column remains at a standstill. This steady rise and fall of the mercurial column is due to the use of very heavy rubber tubing. This instrument is especially adapted for office work.

If desirable, however, to note diastolic pressure, a special indicator, known as the Fedde Diastolic Indicator[8] (Fig. 16) has been devised for the purpose. It is a component part of the new Faught Standard Sphygmomanometer and is described as follows:

The metallic chamber is contained in the base of the case, below the working parts of the instrument, and is attached by elastic tubing to the base of the tube communicating with the glass tube of the indicator. One nipple at the base of the indicator serves for attachment of the tube to the cuff, and a short piece of rubber tubing connects the other nipple with nipple *D* of the sphygmomanometer. The systolic reading is made as usual by obliteration of the pulse, noting the point of its return. From this time attention is paid only to the height of the mercury column, and the motion of the pith ball, synchronous with the pulsations of the artery. As the pressure in the system gradually falls, the pith ball will make an increasingly greater excursion which, when the maximum is reached, will suddenly become small again. At the moment of this change, the diastolic reading is made.

A is the inner arm-band of soft rubber through which the compression of the vessel is obtained.

B is the outer retaining inelastic cuff which serves to prevent the loss of pressure outwardly and which is held in place by means of two straps with friction buckles.

C is a reinforced rubber tube joining the arm-band to the apparatus at the nipple *D*.

P is the piston pump which furnishes the source of pressure and which is connected to the nipple *F*, bearing a stop-cock *M*, whose function is to eliminate elastic pressure of the pump and tube during the diastolic reading.

[8] Made by G. P. Pilling & Son Co., Philadelphia, Pa.

N represents a needle valve by means of which the pressure within the pneumatic system of the apparatus is maintained or regulated.

H is the manometer tube, and *K* and *L* are the mercury guard cocks.

G is a specially devised adjustable scale, which gives a reading directly in millimeters of mercury.

Dr. George Bachmann (*New York Med. Journ.*, 1911, XCIII. 212-215) has described a new aneroid instrument devised by Pachon of Paris and known as the sphygmometric oscillometer (*Comptes Rendues de la Societê de Biologie*, 1909, LXVI. 733-735), which has been designed with the idea of overcoming the errors seen in instruments of the Riva Rocci type, on the theory that mercurial

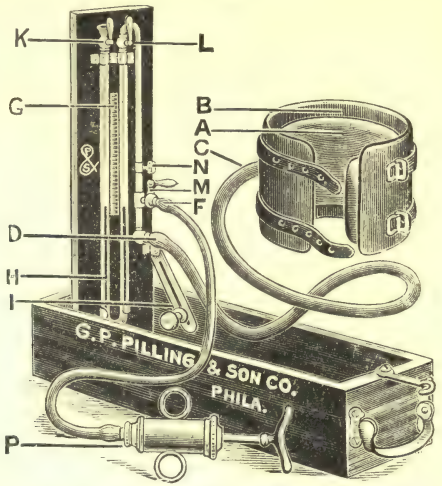

Fig. 16—New Faught Standard Sphygmomanometer.

manometers are not sensitive enough to demonstrate the amplitude of oscillations in noting diastolic pressure. The essentials of this instrument (Figure 17) are a metallic box, hermetically sealed, and containing in its interior an aneroid dome connected with a lever. *M* is an aneroid manometer which indicates the level of the arterial pressure. Box, manometer, dome, and cuff are connected by tubes. The pressure can be raised by the pump or lowered by allowing the air to escape at *S*. The cuff and dome can be cut off from each other. The armlet is adjusted as usual, then air is pumped in until the manometer registers a degree of pressure above ordinary arterial

pressure. The valves are then manipulated until the level *N* denotes an excursion of about one degree on the dial. This indicates the return of the pulse at the compressed point, and therefore the maximum systolic pressure, which is now read on the manometer *M*. To determine the minimal diastolic pressure, the air valves are again manipulated until the largest excursions of the lever have been reached, when the manometer *M* records the diastolic pressure. In this respect Bachmann thinks the Pachon the most satisfactory instrument for determining diastolic pressure.

Of the many sphygmomanometers now in use, three by physicians of this city, may be mentioned. Stein's[10] spring sphygmomanometer is a small pocket machine, simple and inexpensive. As the spring loses its strength, and is affected by temperature, readjustment of the index is needed from time to time. Bishop's[11]

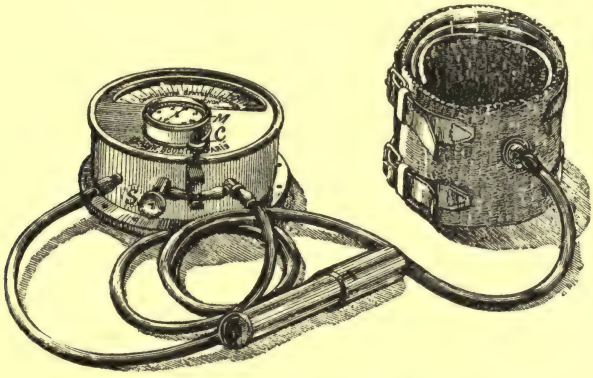

Fig. 17—Pachon's Sphygmometric Oscillometer.[9]

instrument is actuated by water, and is simple and inexpensive; it is adapted for office work rather than for ordinary practice. Bendick's[12] is actuated by air and water. As of the latter only a few c.c. are used, there is little of the inertia inherent in the mercurial instruments.

The following experiments, conducted by Dr. Haven Emerson in my presence, at the physiological laboratory of the College of Physicians and Surgeons of New York, during a recent course in clinical physiology, were done with the von Recklinghausen

[9] Made by Charles Verdin, Rue Linné 17, Paris.
[10] Made by Charles E. Dressler, 1 Madison Avenue, New York City.
[11] *N. Y. Med. Jl.*, 1911, XCIII. 405-407.
[12] *Jl. Am. Med. Assoc.*, 1911, LVI. 1873-4.

instrument, and illustrate some of the uses to which the sphygmomanometer may be applied:

A healthy man lying on his back had an arterial pressure (maximum) of 124 mm. of mercury; when his lower limbs were raised by an assistant so as to be vertical, the pressure rose to 130 mm., and to 145 when they were raised by the man's individual efforts. The effects of posture in blood pressure were thus shown, and the danger of such movements to an arteriosclerotic made very evident.

In a further experiment where a man was directed to strain at stool, his normal (maximum) pressure of 124 rose promptly to 160, suggesting further that in arteriosclerosis the increased pressure may easily superinduce hemorrhage. This recalled to my mind a case of my own, where a lady with tuberculosis brought on a fatal hemorrhage by straining at stool.

The action of certain drugs that affect the circulation was also well shown by the same instrument. Five minims of a 1-1000 solution of adrenalin injected into a vein of the arm caused a rise of pressure in 40 seconds, but the fall within the minute showed how transient is the effect of this drug, when used as a circulatory stimulant, and how necessary it is to repeat such an injection at very brief intervals in order to maintain the initial rise. The effect of the drug in this case was also seen to be violent and distressing.

As showing the difference in pressure, by the subcutaneous injection of the same amount (5 min.), the blood pressure was maintained at about the same level from 2-6 minutes, and with much less disturbing effects.

The inhalation of ammonia was also shown by testing it on the same subject, to raise the pressure 4 mm., while stimulation of the branches of the 5th nerve by faradization of the nares put up the pressure 10 mm.

In line with these experiments are those of Dr. H. W. Cook done with his modification of the Riva Rocci instrument (*Journal of the American Medical Association,* 1908, L. 676-679) when he demonstrated the effect produced on blood pressure by one grain of sodium nitrite used by him in hypertension from vaso-dilatation in a case of cardiac hypertrophy with nocturnal oppression. In this case he found that the action of this drug began in three to five minutes when administered by the mouth, and in about two minutes when given hypodermatically; its ac-

tion was maintained from one to two and a half hours, the full return to the normal tension requiring from two to three and a half hours. On the other hand, he found by using the same instrument that the effect of nitroglycerine did not last more than one hour. As a result of these experiments, therefore, he naturally advises the use of sodium nitrite rather than nitroglycerine when it is desirable to get a more lasting effect.

In a series of three cases, in the service of Dr. R. C. Kemp, in the Manhattan State Hospital, during the spring of 1908, the effects of acute dilatation of the stomach as reducing blood pressure were well shown by my own Riva Rocci instrument.

In each case the stomach was first mapped out. Then while lying on the back, the patient's pulse, respiration and blood pressure were taken. Next the stomach was artificially dilated with tartaric acid and sodium bicarbonate in solution. The evolution of carbonic acid gas almost immediately dilated the stomach, as shown by percussion, while contemporaneously the pulse and respiration rose and the pressure fell.

Here again the use of the sphygmomanometer illustrates the danger to a weak heart from a dilated stomach. Experience has taught me that in from six to eight per cent. of the sudden deaths in cardiac disease that have occurred in my practice, the immediate cause was attributable to some gastric disorder. Gastric dilatation from gas, fluid, undigested food, or a mixture of these is always a source of danger in the chronic cardiac diseases of persons who have passed middle life, and especially in the aged.

In a brief resumé of blood pressure work by Francis A. Faught, the inventor of the sphygmomanometer which bears his name, he calls attention to the increasing use of the instrument in diagnosis, prognosis, and treatment. Owing to the many physiological factors causing variations of readings in the same patient, and the different readings of different instruments, the adoption of standard figures for the individual in health and disease is impossible. Age is an example of a disturbing physiological factor. But taking the normal pressure at 20 as 120 mm. of mercury, he would add half a millimeter for each year above 20. Accordingly, at 25 the normal pressure should be 125; at 40, 130. He finds the widest application of the blood pressure test in cardiovascular and renal diseases; also in myocardial diseases, as a test of the efficiency of treatment in cardiovascular diseases, and in the toxemias of pregnancy.

Conclusions.—(1) The sphygmomanometer has two uses: one for the physiological laboratory or hospital, the other for ordinary practice. Instruments that may be used advantageously for one purpose may not be applicable for the other. Even in ordinary practice one should be guided in the choice of an instrument by the degree of accuracy required; the facilities available for keeping it in good working order; also its portability.

(2) The von Recklinghausen is a good example of a laboratory instrument, the Faught of a clinical.

(3) No physical examination is complete without a record of the blood pressure. It is also very helpful in the diagnosis and management of cardiovascular and renal diseases and toxemias.

(4) In experimental medicine the sphygmomanometer is especially useful in the pharmaco-dynamics of cardiovascular remedies; also in determining the effects on the circulation of altitude, posture, physical exercises, baths and diet, etc.

(5) No one of the sphygmomanometers is absolutely accurate. The errors in estimating maximal pressure vary from 3 to 20 mm. or more of mercury. The diastolic readings have a greater error. But by taking the mean or so-called *pulse pressure* in such cases, the errors may be materially reduced.

(6) When considerable variations are taking place in the blood pressure, as often happens for various reasons, several tests should be made at longer or shorter intervals of the systolic alone or of systolic and diastolic in association, according to the degree of accuracy required. An average of these readings constitutes the so-called *pulse pressure*, the best criterion of pressure available at present.

CHAPTER III.

GRAPHIC METHODS AND INSTRUMENTS IN THE DIAGNOSIS OF CARDIAC AFFECTIONS.

Graphic methods are used in medicine to record tracings chiefly of cardiovascular, respiratory, and muscular movements.

Polygraphy is a method of recording two or more tracings simultaneously on kymographic paper actuated by a motive force, usually clockwork or electricity. The paper is attached to the surface of a metal drum, or sometimes to two drums. Of the tracings one may be made to mark the intervals of time in seconds or fractions of seconds. Kymographic paper is usually white with a smoked surface, the tracings made by the pen-arm or stylet revealing the paper and so appearing as white. This method records the precise length of a cardiac cycle, the several events being registered in waves or depressions. By this means a number of features of the circulation that were formerly unknown to us are disclosed. The tracings are truthful records of events, subject, however, to errors produced by defective instruments, sometimes by artefacts that cannot always be avoided, and also by lack of skill on the part of the operator. So, too, where there is an unusual combination of cardiac arrhythmias, even an expert may be in doubt as to the proper interpretation of each wave, wavelet, or depression.

But despite these drawbacks, tracings are fairly comprehensible registers of the various cardiovascular activities. In fact, polygraphy can be used effectively to determine the action of drugs, food, and drink on the human organism, and also such other therapeutic agencies as baths, muscular exercises, massage, and electricity. As will be shown, it is also a material aid in the diagnosis of cardiac disease.

In arrhythmias it has led to a new classification. At the present time polygraphy is the most reliable guide we have in determining abnormal cardiac conditions. It is therefore helpful in indicating appropriate lines of treatment.

But the sphygmograph alone is of comparatively little value, because there has never been any close agreement among physiologists and clinicians as to all the characteristics of a normal sphyg-

mogram, nor as to distinctive curves in the various forms of valvular disease. This conclusion I reached as early as 1882, when I took the matter up at the Presbyterian Hospital of New York City. Though at that time most of my colleagues were of the opinion reached by Rosenstein in 1876, that no differentiation in valvular diseases can be made by any sphygmograph, a single one of them clung to the notion that aortic regurgitation had a distinctive style of curve. My experience showed me at that time that even on this point such an opinion was erroneous. Sphygmograms are also lacking in accuracy because the personal equation cannot be eliminated. To secure a good tracing the exploratory button or base must be placed directly over the radial, so as to press it against the bone. This is not easy. Besides, a certain, but indeterminate degree of pressure must be employed, and, inasmuch as it is impossible to adjust the button precisely in the same spot or to use exactly the same amount of pressure in each instance, the tracings will vary in successive tests. Indeed, there is no such thing as a standard normal sphygmogram. Age, the degree of vitality, and sex are some of the factors that cause variations in the sphygmogram in health. Similarly, there can never be any fixed standard for the tracings of the various diseases of the cardiovascular system. As each individual differs from another in health, so, even in the same disease, and in successive examinations, there are differences which will be recorded in the sphymogram. One has but to compare the several tracings in health and in valvular diseases as given by Eichhorst, Colbeck, Mahomed, Strümpell, Fagge, Michael Foster, and others to assure himself that my statement is substantiated by their experiences. Even in a single examination, the tracings will show appreciable differences, as will be shown in Fig. 19. The same statements are applicable to the cardiogram, where the variations are even further emphasized.

Yet, the sphygmograph has its uses. In fact, we cannot well dispense with it, even though its field is comparatively narrow. It may be relied on to give the frequency of the pulse, when the finger is unable to count it. It will give a rough record of some cardiac arrhythmias. It is competent, moreover, to demonstrate auricular fibrillation without any other appliance, if we rely on recent experimental researches.[1] But it will not indicate the grade and quality of arterial tension as well as the trained finger. As a

[1] Lewis.

key, however, to the interpretation of the jugular pulse, its tracings are most important, and in simultaneous records of the jugular, carotid, and radial pulses and the apex beat the sphygmograph applied to the radial affords a better criterion than the carotid, because it is more easily isolated from the surrounding tissues.

In the field of polygraphy, we must, for the present at least, be satisfied if its records give us practical assistance, even if they differ. Their accuracy is comparative. The case is much the same as that of the sextant which the captain of a ship uses to find his position when out of sight of land. He cannot, except by the merest chance, determine the real position of his ship, for he has no stationary mark to assist him, but he nevertheless usually locates his position on the chart with a sufficient degree of accuracy for practical purposes. The analogy holds good with many of the instruments in use in medical practice, but they assist us materially in framing our diagnoses. Of all the graphic instruments the electrocardiograph is the most accurate, but the character of its curves varies according to the so-called "leads" that are used,—a point that is now generally recognized.

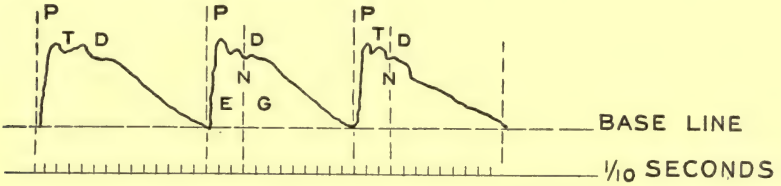

Fig. 18—Schematic plan of a normal sphymogram.

In Fig. 18 is shown the scheme of a normal sphygmogram in a healthy man with a pulse of 72. The space *E* marks the period of ventricular systole, which in this instance occupies about 3-10 second. This is the sphygmic, or pulse, period. The space *G* marks the ventricular diastole, and occupies about 9-10 second. The nearly vertical upstrokes *PP* are known as the percussion waves; the tidal waves *TT* follow. At *DD* are seen the dicrotic waves, while *NN* represent the dicrotic notches. An imaginary line runs horizontally through the lowest points of the upstrokes, and is known as the base line, while under it the intervals of time are marked by the chronograph in fractions of a second. The waves, wavelets, and notches and their time relations to one another are of great importance in deciphering the significance of a jugular tracing such as is seen in Fig. 19.

As far as possible, both the numbers and letters used in these illustrations will be those of the English system, as used by Lewis. Unfortunately, there has been no uniform system of notation, and much confusion has resulted. The accepted intervals of time in Figs. 18 and 19 are those of Mackenzie, who makes the cycle occupy 1200-1000 seconds, while Michael Foster puts it at 1130-1000 seconds. The latter observer puts the duration of ventricular systole at 451-1000 and the duration of ventricular diastole at 679-1000 second. Lewis puts the length of the cycle at 1010-1000 seconds, the ventricular systole at 540-1000 second, and the ventricular diastole at 470-1000 second. Of course, the length of the cycle varies with the frequency of the pulse. If a pulse of 72 has a cyclic length of 12-10 seconds, a pulse of 60 will have a cyclic length of 10-10, or 1, second. More than this, and as a corollary to what has already been said, there is no absolutely fixed relation in time between the periods of ventricular systole and the diastole of either ventricle, carotid, or radial, as may be seen by measuring these intervals in Fig. 19 with a pair of dividers.

In Fig. 19 the carotid shows a nearly vertical upstroke, due to

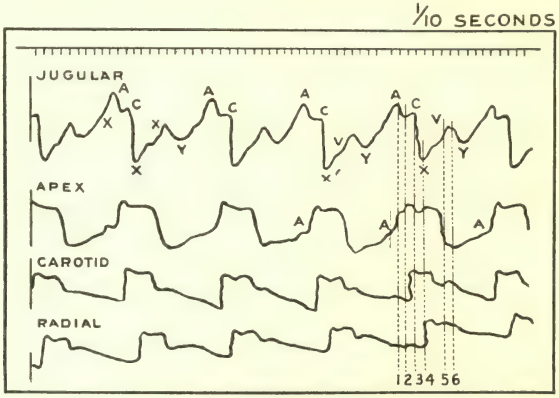

Fig. 19—Simultaneous tracings of jugular, radial, and carotid pulses and apex beat. An adaptation of a Mackenzie polygram in a healthy man with a pulse of 72, where the length of the cardiac cycle is $\frac{10}{12}$ seconds.

the sudden rise of blood-pressure caused by ventricular systole. It is followed by a long and irregular downstroke, due to the gradual fall of blood-pressure. The first or tidal wave is due to secondary contraction and expansion of the artery immediately after its primary systolic contraction. The second wave, called the recoil or dicrotic wave, is caused by the recoil of the blood column due to the closure of the aortic valves. The carotid upstroke precedes the radial upstroke from 1-10 to 2-10 second.

In the jugular pulse, *A* is the auricular wave, *C* the carotid wave, and *V* the ventricular wave; *X* is the carotid depression and *X'* the auricular depression, while *Y* is the ventricular depression.

The numerals 1-6 refer to contemporaneous events in the four tracings, so that their effects can be plainly observed in each case. At 1, auricular systole is seen in the jugular. At 2 systole begins in the ventricles. At 3 the aortic and pulmonary valves open. At 4 the radial is seen to be contracting about 1½ seconds after the carotid. At 5 the aortic and pulmonary valves are closing. Between 1 and 3 is the interval between the beginning of auricular systole and the opening of the aortic valves. It is known as the *A-C* interval.

Apart from the two normal oscillations of the downstroke, as seen in the radial and carotid, there are other miniature waves, some of which are referable to the inherent elasticity of the arterial walls, and some to instrumental or other causes, such as auricular fibrillation. It will be seen that the apex of the normal arterial tracing (Fig. 18), or the angle between the upstroke and downstroke, is nearly that of a right angle, while of the two minor downstroke waves the recoil is more conspicuous than the tidal. This angle in many cases is quite acute, as may be seen in Fig. 18.

The cardiogram, shown by the tracing of the apex of the ventricle in Fig. 19, consists of a nearly perpendicular upstroke, a nearly horizontal line, the systolic plateau (3-5), and an oblique downstroke (5-6). The upstroke marks the beginning of ventricular systole, and may be preceded by a minor wave (*A*) due to systole of the left auricle. Ordinarily the cardiogram fails to show this auricular undulation, but faint indications of it may be seen in the radial tracing of this polygram. It is usually well shown in the electrocardiogram. The sloping line of the apex tracing (5-6) is often rippled by other subsidiary waves. Chronologically, the systolic plateau corresponds to the impact of the heart against the parietes during ventricular systole, and from this summit the downstroke falls with moderate obliquity to the base line. The wave following immediately after the downstroke (5) coincides with ventricular diastole.

The phlebogram, as illustrated by the jugular tracing in Fig. 19, is composed of three distinct waves. The first of these, the auricular, or *A*, wave, is presystolic in time, being coincident with the contraction of the auricles. The second wave (*C*), commonly called the carotid, is to be attributed to the communicated impact of the

carotid artery. The third wave (*V*) is known as the ventricular; it corresponds in time with the dicrotic wave in the radial. The notch after the *A* wave marks relaxation of the auricle; that after the *C* wave denotes auricular diastole; that succeeding the *V* wave indicates ventricular diastole and the passive period of the cardiac cycle. The *A-C* interval is the interval between the beginning of the auricular systole and the opening of the aortic valves, typified by the carotid waves in the tracings of the jugular pulse. (Figs. 19, 21 and 22.) This *A-C* interval is in health about 1-5 second.

When there is the ventricular type of jugular pulse, the jugular pulse corresponds in time to the systole of the ventricles, *i.e.*, venous systole and venous diastole are contemporaneous with ventricular systole and diastole. There is in such cases a change in the point of origin of the cardiac stimulus; indeed, where auricles and ventricles beat simultaneously the impulse probably originates in the ventricle. (See Fig. 23.) Contrary to opinions that have heretofore been expressed, the venous pulse can usually be found and registered, though the method is not always easy (Barringer). Anything that produces increased venous pressure, such as intrathoracic tumors or abdominal pressure, is likely to cause prominence of veins such as the jugular. The causes, however, are not all known.

Fig. 20, adapted from Lewis, gives a schematic representation of the waves and depressions of the carotid, aortic, ventricular, auricular, and jugular beats, as compared with the electrographic tracing. The several curves are a compound made up of many tracings in individuals whose pulse is set at 60. The length of the cycle is set at 1 second. It will be noted that in the auricular and jugular tracings there is much variation as to the nature of the waves; also that in the electrocardiogram both the waves *R* and *T* anticipate in time and auricular and ventricular waves as seen in the ventricular tracing. It is supposed that the electrogram registers the contractions of the papillary muscles, which precede those of the main part of the heart wall.

Now, the cardiogram, which is the record of the apex beat, taken either immediately over the seat of the visible impulse, in the fifth interspace or in its vicinity; in the second, third, or fourth space, or in the epigastrium, helps us also to interpret the venous pulse tracing. The cardiogram can, besides, indicate whether the left or the right ventricle makes the impact, for when the right apex makes the impact the tracing is inverted.

This illustration gives a fairly correct view of the contemporaneous happenings in the different cycles, though, as has already been said, there is such a variation in any individual in the length of the cycle and in the prominence and position of the waves and depressions that no one of the tracings can be considered as more than approximately correct.

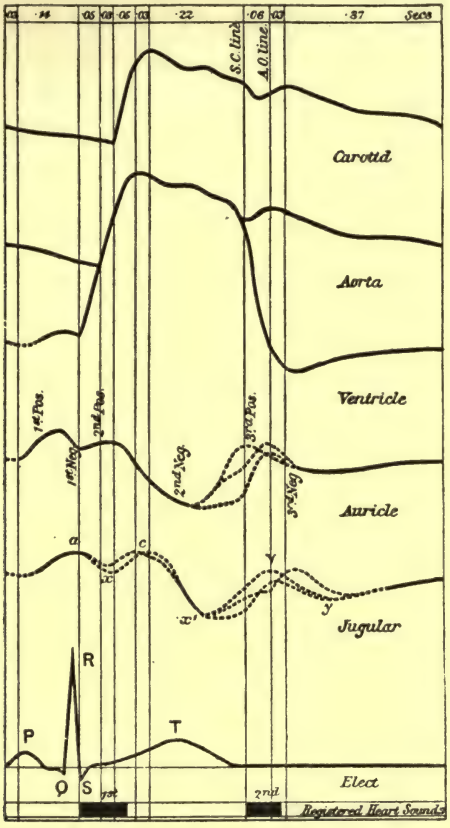

FIG. 20—Diagrammatic representation of tracings taken simultaneously.

When the apex beat is not clearly felt, a tracing of it may sometimes be obtained in the epigastrium, but it may be necessary to put the patient in the sitting position. If the beat is of the right apex, the cardiogram will be inverted. It will be seen, therefore, that records may be made of the action of both right and left ventricles; the phlebogram, however, gives the record of only the right ventricle and right auricle, while the sphygmogram gives the record

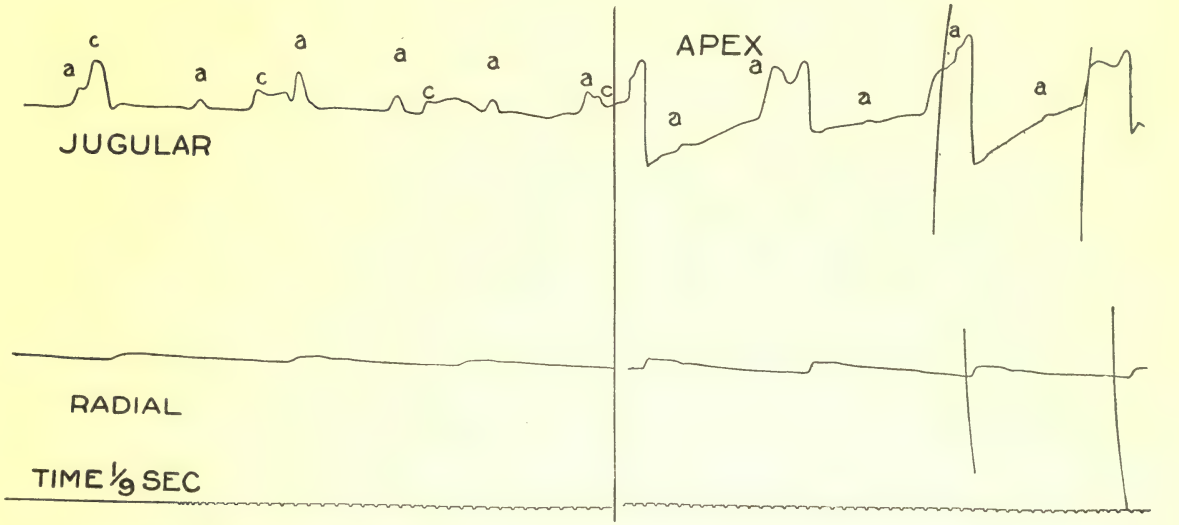

FIG. 21—In this illustration is seen on the right the tracing of the apex (left ventricle). The waves at *a* denote the impression recorded on the tracing by the contraction of the auricles. The case is one of complete heart block, the ventricles and auricles operating quite independently of each other. This tracing was taken immediately over the point of cardiac impact by Dr. T. B. Barringer, Jr.

of both the left ventricle and left auricle. Polygraphic methods thus give a record of events in the four chambers of the heart.

In Fig. 5, a tracing taken by the author, it will be seen that no two of the successive apex beats have the same length. This is, therefore, according to the criterion of Lewis, an example of auricular fibrillation, the so-called "nodal rhythm" of Mackenzie, or the permanently irregular pulse of Hering. It may occur with the frequent pulse, also, when, in my experience, the prognosis is more grave. It will also be noted that in 12 seconds only one-half the beats have any considerable degree of force. In the jugular tracing, which is not shown, auricular systole was contemporaneous with the apex beat, showing the ventricular type of auricular systole.

FIG. 22—Auricular fibrillation with partial heart block in a male with Adams-Stokes disease. Pulse 30. Time markings in seconds and tenths of seconds.

In Fig. 23, taken from Mackenzie, is seen a radial tracing above, while below is a combined jugular and carotid tracing, the carotid being the dominant type; the impulse of the jugular indicating auricular contraction is transmitted to the carotid in the wave *a*. Here for every 6 beats of the carotid there are 11 of the auricle; the ratio of auricular to ventricular pulsation is therefore as 11 to 6.

In interpreting the jugular pulse we should remember that sometimes the carotid is mistaken for the jugular, so that the two are traced jointly, though in this case the carotid gives the dominating curves. If the radial pulse is small, and there is a pulsating vessel in the neck, it is almost necessarily the jugular. But the simplest way for a novice is to select someone who has marked prominence and fullness of the jugular, and learn to make tracings with him as a model. Examination is best made when the patient is lying down, when the pulsation is better seen. Still, as a rule it is not visible, the vein being covered by the skin, the sternomastoid, and more or less fat-tissue.

The method of analyzing the jugular tracing in a normal radial pulse of 72 is as follows (Fig. 24): Make a downstroke at right angles to the timemarking line, at the beginning of the radial up-

stroke marked 4. Then draw another vertical line 1-10 second or so in advance of it, so as to pass through the top of the middle jugular wave of the three in series. The carotid wave, as we have seen, anticipates the radial by 1-10 to 2-10 second, and corresponds to the middle wave; in fact, it makes this wave. It is, accordingly, to be marked C. Now measure the distance from the beginning of the tracing to 3. Measure a like distance off in the jugular, and it should strike the top of the middle wave. This is a corroborative

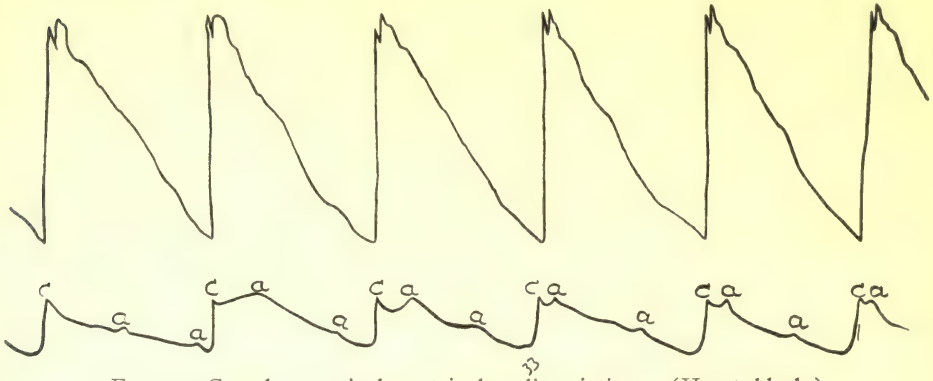

FIG. 23—Complete auriculoventricular dissociation. (Heart block.)

indication that the letter *C* is correctly placed. But the auricular systole occurs about 2-10 second earlier. Draw a vertical line about 2-10 second further in advance; let the line pass through the crest of the wave, and the wave of auricular systole is thereby located. Mark it *A*.

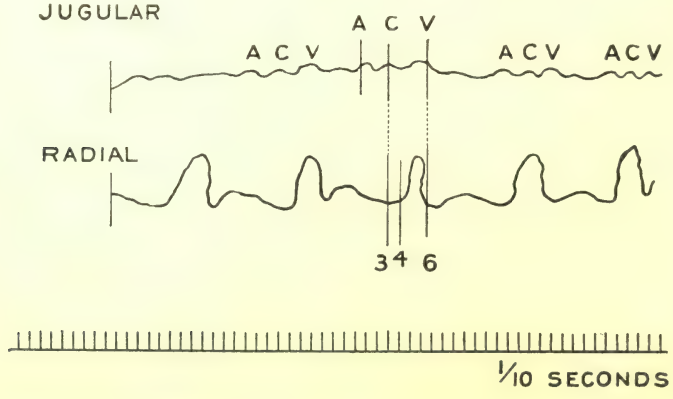

FIG. 24—Method of interpreting the jugular pulse.

The dicrotic notch, which corresponds approximately to the top of the third jugular wave, marks the closure of the tricuspid and mitral valves. Mark this point with the letter *V*.

These three letters are the keys to the interpretation of the jugular pulse; the letters indicating the depressions as shown in Fig. 20 can be added if need be.

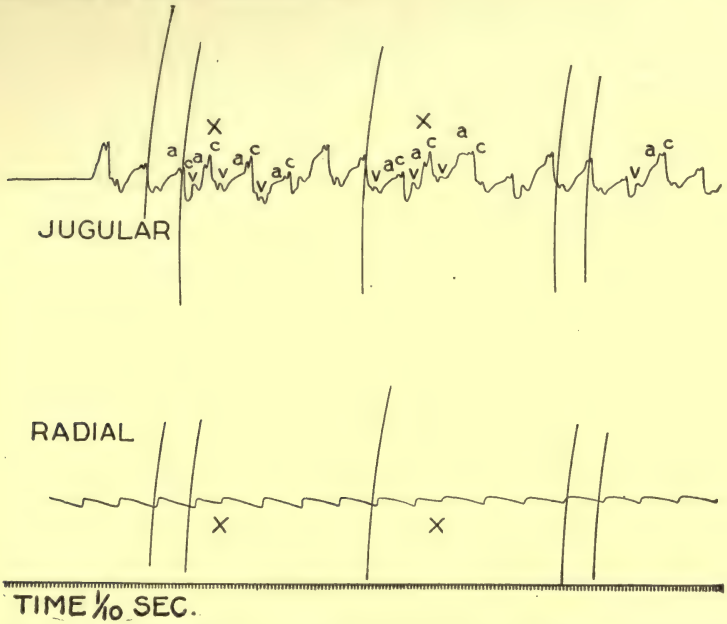

Fig. 25—Extrasystolic arrhythmia of the auricular type. The letter *a* indicates auricular contraction of the right auricle; *c* the carotid wave, and *v* the ventricular wave. At × are extrasystoles of the auricular type. Tracings taken by T. B. Barringer, Jr.

In Fig. 25 the sign × denotes extrasystoles. As already stated, the record of the venous pulse discloses more features than a record of the arterial pulse.

The ordinary polygraphic machine is fitted with one or more delicate levers, each tipped with a stylet, and attached to a tambour with rubber membrane connected by a rubber tube with a receiver, which, when placed over the pulsating area, transmits the undulations to its stylet. The lever, with or without the tip or stylet, forms the so-called pen-arm, and is made to rest lightly on the surface of the kymographic paper, which is usually smoked. As the paper is actuated by clockwork, or in the laboratory by an electric motor, it moves at uniform speed, while the oscillations of the pen-arm on the carbonized film make the graphic tracing.

In order to interpret the several tracings of the polygram, a chronograph, or time-marker, is necessary. This also is actuated by clockwork or electricity. After the polygram has been made, it is to be suitably labeled with the name of the patient, the date, and the locality. Afterward, the numerals and letters required for purposes of interpretation are inserted in their appropriate places. Next, the paper is carefully removed from the drum or drums, and immersed in a solution either of shellac and alcohol or of benzoinum

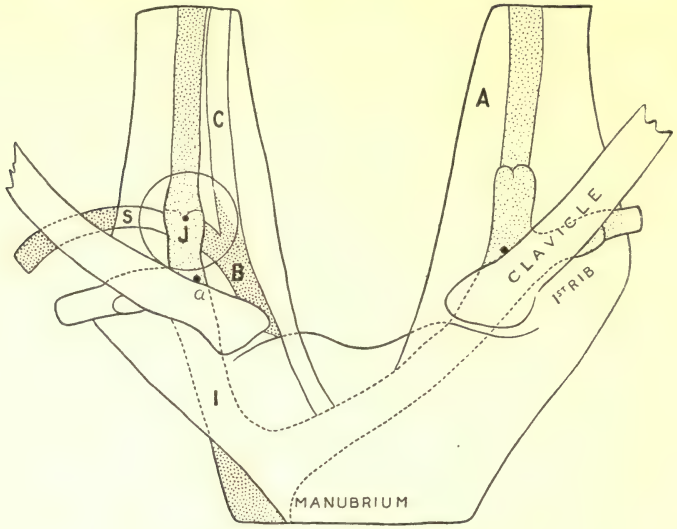

Fig. 26—How to get the jugular pulse.

and alcohol (1 oz. to 10 oz.), after which it is hung up to dry, and then laid on a flat surface, protected from the dust. This is the best method to follow, as unless it is spread out flat before becoming fully dry the paper is apt to curl up in drying.

Mackenzie recommends two instruments for clinical work: the clinical polygraph and the ink-writing polygraph. Both are portable. In hospital and laboratory work, larger and more complete instruments are necessary; these are, of course, more accurate, but their size and weight make them too bulky for ordinary clinical work.

When the tracing is about to be made, the patient should, as a rule, be placed in a comfortable reclining or horizontal position, with the head bent slightly on the chest. Then the operator marks with a dermographic pencil the site of the right radial artery, just above the styloid process of the radius. The mark should be placed where the vessel is most prominent. The wrist is put at rest in an

easy position, and the pad of the machine applied to the spot marked. The rubber tube is then attached to the lever, the tip of the pen-arm is approximated to the surface of the smoked paper, and the spring connected with the lever is so regulated as to get the required amplitude for the excursions of the pen-arm.

To get the jugular pulse, apply the receiver,—which is a brass cup, perforated with a minute hole to allow the escape of air when applied,—over the jugular bulb on the right side, at the spot (*J*) indicated in Fig. 26 (after Mackenzie).

Occasionally it may be better to apply the receiver on the left

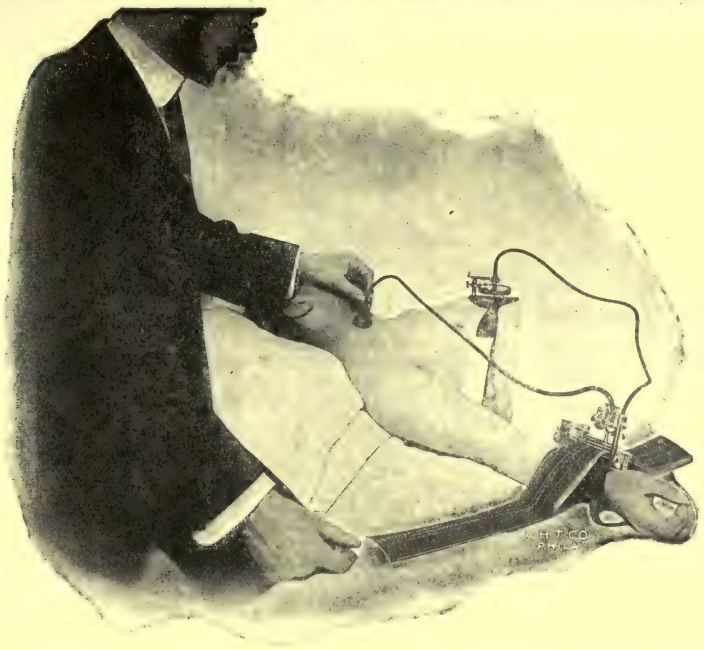

Fig. 27—Taking a phlebogram, cardiogram and sphymogram simultaneously.

side or higher up. The other end of the tube should be attached to the pen-arm and approximated to the kymographic paper.

In getting the liver pulse, a special receiver is required. It should be large and oblong. After the knees of the patient have been drawn up, it is applied to the surface of the liver, being pushed up under the free border of the ribs. To this receiver one end of the rubber tube is attached and the other to the pen-arm and tambour.

A somewhat similar receiver is placed over the apex of the

heart, where it is bound down by a circular band (Fig. 27). All of the pen-arms are firmly attached to a vertical metal support, which is firmly clamped to a table, bracket, or some other object that is immovable.

To record the carotid pulse, the receiver is placed at about the level of the thyroid cartilage, or at the level of the hyoid bone, on the right side. The chin should be elevated and the head turned to the left, in order that the carotid may be easily reached by the receiver.

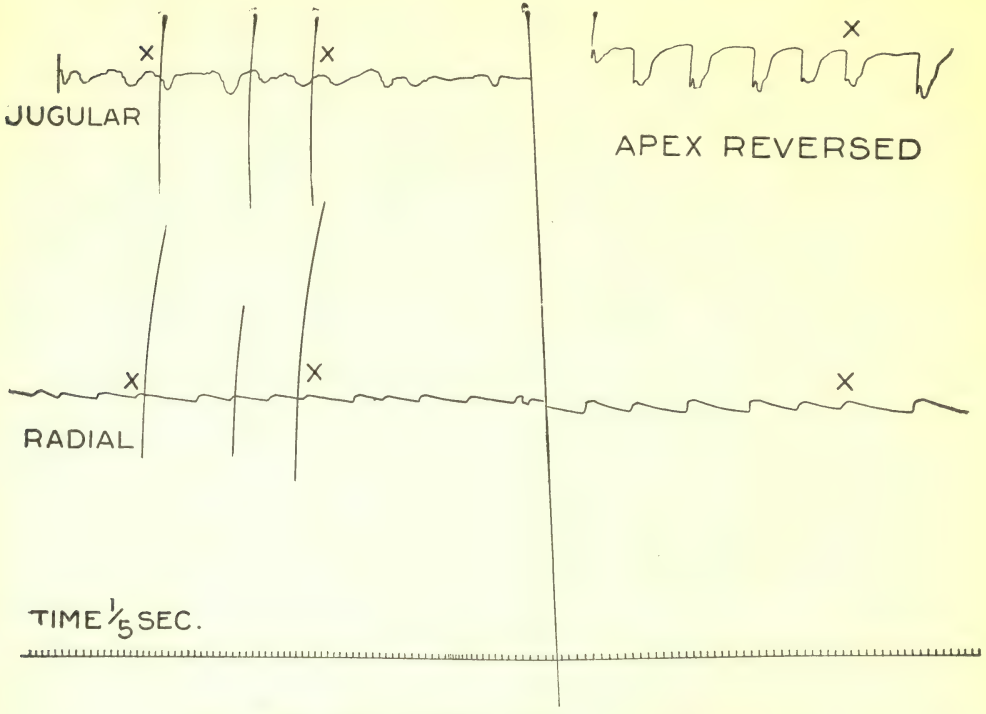

Fig. 28—Reversed apex beat. By T. B. Barringer, Jr.

It was thought that the venous pulse could not always be taken, but Dr. T. B. Barringer, Jr., of New York, was able to take it in 25 successive young persons with normal hearts.[2] The venous pulsations may best be seen in the neck, when the patient is lying down and the receiver is applied about an inch and a half from the sternoclavicular joint, just above the clavicle, over the site of the

[2] Dr. Barringer made them run up three flights of stairs, and then used a rather shallow receiver of a special type.

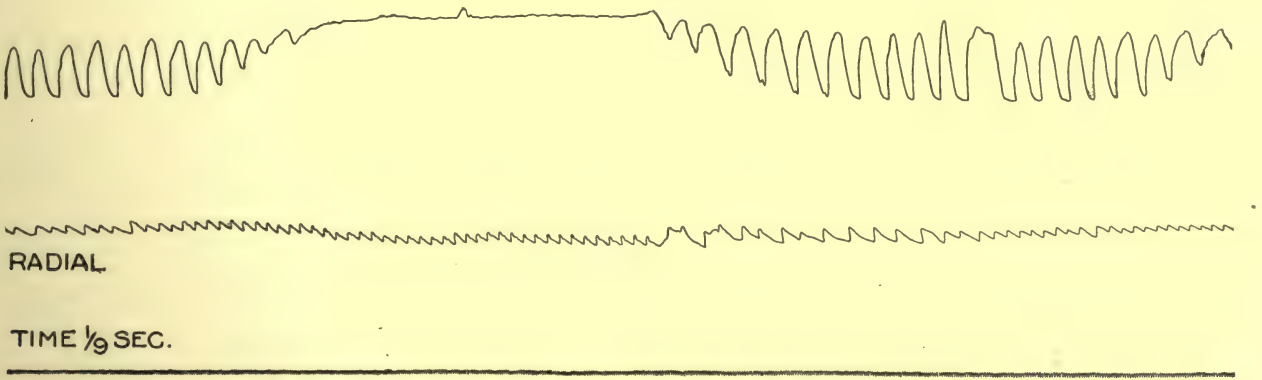

FIG. 29—Cheyne-Stokes respiration. By Dr. T. B. Barringer, Jr.

jugular bulb. But the right sternomastoid must be in a state of relaxation, which necessitates such a position of the head that the tension of the muscle is overcome. If the respiratory movements are too prominent in the jugular tracing, it will be advisable to have the breathing stopped for a while, so as to eliminate this feature.

If, in taking the record of the apex beat, the receiver is placed on the inner side of the apex beat, we may get the record of the impulse derived from the right ventricle, which will make an inverted tracing as shown in Fig. 28.

In the obese, and in women with pendulous breasts, there may be no visible apex beat. In pronounced myocardial diseases it is apt to be absent. Whenever the cap or receiver is used, the finger must cover the minute air hole while the tracing is being made, so that the full force of the column of air will be transmitted to the tambour.

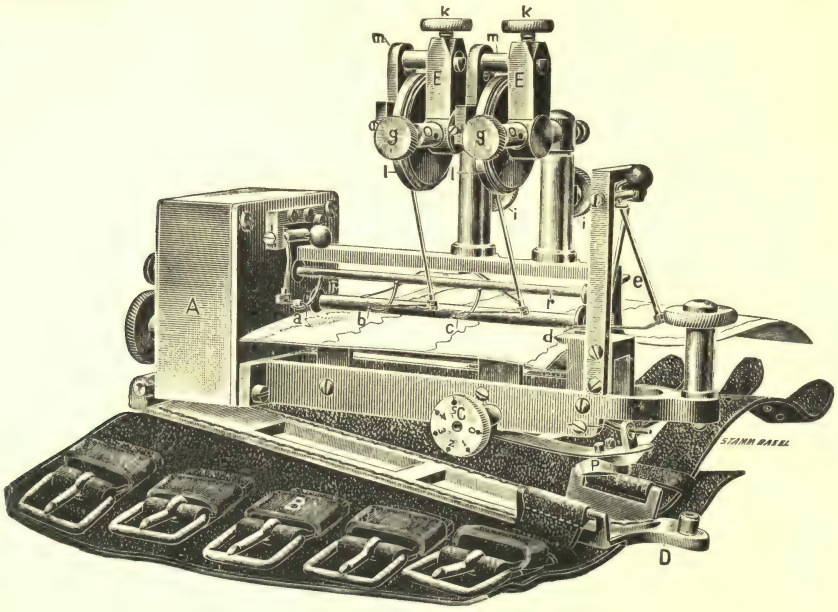

Fig. 30—Jaquet's sphygmocardiograph.

In taking respiratory movements such as are seen in Fig. 12, bind an ordinary rubber bag to the chest, attaching to it a tube and tambour; the respiratory movements will be traced on the moving smoked paper.

The Jaquet polygraphic machine, known as the sphygmocardio-

graph, is excellent for clinical work, though its cost in this country, duty paid (Arthur H. Thomas Company, Philadelphia), is $130. This instrument is compact, comparatively easy to operate, and capable of registering three tracings. Though these are miniatures of the tracings made by large laboratory kymographs, they give the prominent details.

Jaquet's sphygmocardiograph (Fig. 30) is provided with a small metal plate which rests on the subject's radial artery, and is attached to a lever system carrying at its free end a delicate stylet for registering the movements of the radial pulse. A second stylet and lever system plays upon a tambour, and leads by a rubber tube to a special receiver designed for the cardiac apex or other thoracic pulsations, and held in place by a chest strap. A third registering mechanism of similar construction communicates with a cup-shaped receiver used for transmitting the jugular impulse; a fourth, actu-

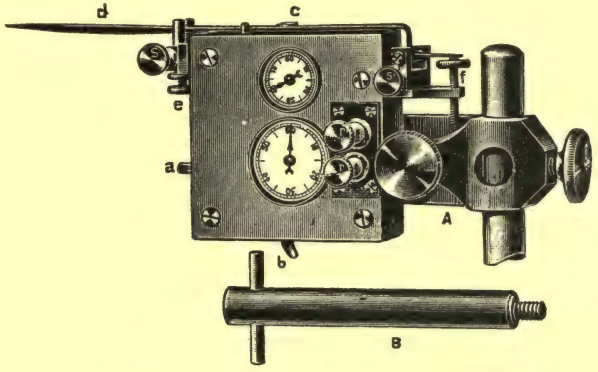

Fig. 31—Jaquet chronograph.

ated by separate clockwork, marks the time. When, after adjustment, the three stylets rise and fall with proper amplitude, indicating that the different undulations will be satisfactorily registered, the operator starts the chronograph and sets the strip in motion, adjusting it to run its whole length, while an assistant catches the paper as it passes from the rolls and guide wheels, so that it emerges without hitch from the instrument.

Marey's polygraph is bulky, but accurate; its cost is $120, duty paid.

Gibson's polygraph takes four simultaneous ink tracings on glazed paper, but it more expensive than either of the two already mentioned. There are also numerous other instruments, such as those of Dudgeon, Frey, Richardson, and Mackenzie.

One of the best time-markers is the Jaquet, shown in Fig. 31. It can be used in connection with any polygraphic machine. When the pen-arm (*d*) is applied to the surface of the kymographic paper, it will record time tracings with intervals of seconds and fifths of seconds. It is operated by clockwork, the dials of which are shown in the cut. When the instrument has been attached to the vertical rod at *A* and clamped in position to the screw, pressure is made on the button *b* for seconds and at *c* for fifths of seconds, after which the lever is released, and the pen-arm writes the time divisions on the paper.

The best hospital polygraphic machine is that of Dr. T. B. Barringer, Jr.

Chapter IV.

AUTOMATIC POLYGRAPHY:

The Electrocardiograph: the Spring Recorder. A New Electric Time Marker.

THE ELECTROCARDIOGRAPH.

Muscle tissue, in the performance of its functions, evokes three things: (1) Animal action or motion; (2) Animal warmth; (3) Animal electricity. The first of these is, of course, the most important to the economy. As compared with it, the production of animal warmth and electricity are insignificant.

Now, the discovery that electrical currents emanate from contracting muscular tissue was foreshadowed by Harvey when he published his "Exercitatio Anatomica de Motu Cordis," etc., in 1628. But Matteuci in 1843 was apparently the first who actually demonstrated it, confirmation coming from Kölliker and Remak, independently, in 1850. During the last ten years no less than sixty individuals have made a study of it, while, according to a list of references in Kraus and Nicolai's recent book, "Das Electrocardiogram" (1910), no less than twenty-five have written on electrocardiography. The reason for this great activity is to be found in the importance of utilizing electricity for commercial purposes; otherwise, electrographic methods would not have been brought so rapidly to their present standard of efficiency. In fact, it was Ader's registration machine, as applied to submarine cable work, brought into notice in 1897, that led to the construction of the string galvanometer which is an essential element in the electrographic machine. For the one most in favor, made by Edelmann and Son, of Munich, utilizes the Einthoven or string galvanometer. It should be stated parenthetically, however, that Lippmann, in 1873, by his invention of the capillary electrometer, had already furnished the idea of a hypersensitive capillary electrometer.[1]

The idea of registering the action of heart muscle depends upon the fact that there is a ratio between the contracting force

[1] Dr. J. B. Stein of the College of Dentistry has for some years been in the habit of using this electrometer in his demonstrations of cardiac action in the lower animals.

of certain heart muscles and the current that emanates from them. This fact was discovered by H. Waller, of England, as early as 1887. Putting it in another way, electrical currents are to some extent measures of the muscular action of the heart's chambers. This does not militate against another fact, that some muscular contractions are not always shown by electric equivalents on the sensitive paper of the Einthoven machine. For faulty manipulation of the apparatus may, in some instances, explain this omission.

Now, as already said, Matteuci discovered in 1843 that electro-

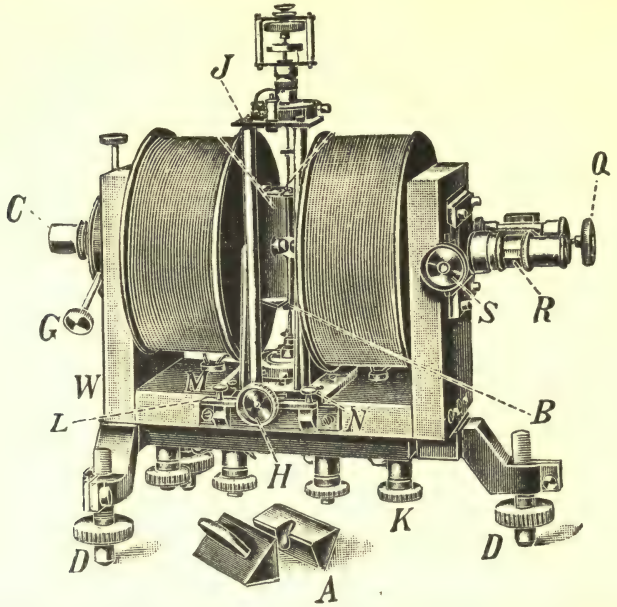

Fig. 32—The Einthoven galvanometer electromagnets and microscope.

motive force emanated autochthonously from the heart. DuBois Reymond confirmed this discovery in 1849 by the use of a very delicate galvanometer, Remak and Kölliker following, as already stated, in 1850. Müller and Kölliker showed later (1850-1856) that there was a special current developed in the auricles during their contraction.

Lippmann, however, in 1873, appears, in addition to his invention of the capillary electrometer, to have been the first to devise a registering apparatus, though Marey of Paris subsequently made a very good recording instrument. The latter was able

to throw the shadow of a moving column of mercury on an open space behind which was a moving sensitive plate upon which were photographed the oscillations of the column. But Marey's work was overlooked. Between 1887 and 1889 Waller was developing his capillary electrometer that registered the heart's action. Altogether it has taken two-thirds of a century to complete the Einthoven machine of the present day.

For the string galvanometer is the standard instrument, and that of Einthoven of Leyden is the one in general use. Ader used at first a capillary galvanometer made of fine copper or aluminum wire which was suspended at right angles to the poles of a permanent magnet. He used a wire as much as 100 cm. long, in order to have larger excursions. The diameter of the wire was about 2/100 of a millimeter.

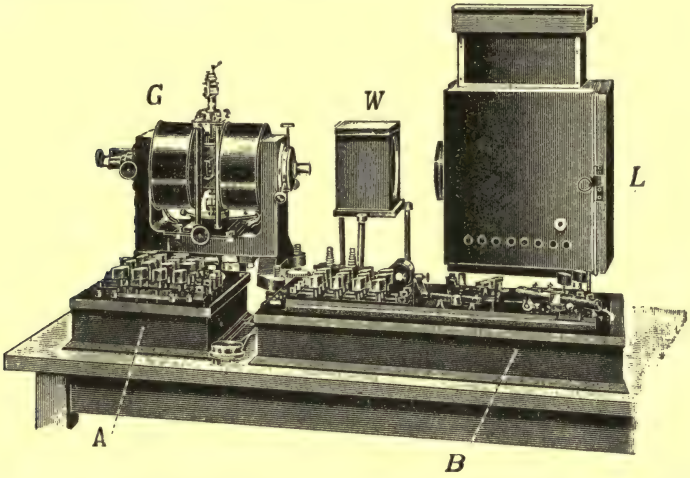

FIG. 33—The Einthoven electrocardiographic machine fitted to a table.

Ader, in 1897 (*Comptes Rendus de l'Acad. des Sc.*, 1897, CXXIV. 1440), devised his instrument, known as Ader's receptor, to supersede those of Thomson (Lord Kelvin), known respectively as the mirror receiver and the siphon recorder. By the mirror method the electric current passed through a large coil within which was a copper tube containing a magnetized needle hung by a short thread before a mirror. The needle oscillated under the action of the currents, and the operator sitting before the mirror read the dispatch as he would read the Morse alphabet. This instrument has, however, been superseded by the siphon recorder,

used very largely at the present time by the transatlantic Commercial Cable Company of this city. It was invented by Thomson in 1867 (*Bright's Submarine Recorder,* p. 630).

The siphon recorder apparatus consists of a very light coil of wire suspended between the two poles of an electromagnet, and capable of turning on its vertical axis. According to the direction of the current, the coil turns one away or the other. The motion of this coil is transferred to a capillary glass siphon, one end of which is bathed in ink while the other is approximated to a slip of paper moved by clockwork. The ink being connected with a small electrostatic machine and the paper with the earth, the ink is attracted to the paper, and issues from the capillary tube with a rapid succession of spurts. When the siphon is at rest it writes a straight line on the moving paper—in this instance a tape—but when actuated by positive and negative currents it oscillates from one side to the other, and the deviations above and below an imaginary line correspond to the dots and dashes of the Morse code. An operator reading the marks on the paper, as they pass by clockwork before him, converts them at once, on the same paper, into the Morse code, while another operator sitting opposite reads the Morse message and simultaneously typewrites it into the ordinary message.

Now, in the Ader recorder a long vertical wire, through which the current passes, is stretched in a magnetic field, and is drawn from side to side by the poles of the magnet, while a ray of light from a lamp throws the shadow of a minute portion of the moving wire on a moving strip of photographic paper in the form of an undulatory dotted line. This method has been used extensively by some of the French submarine cable companies, while the Einthoven machine is an adaptation of it for electrographic work in heart disease.

But Einthoven, the inventor, and Edelmann, the manufacturer, have improved on the Ader galvanometer. Einthoven's is now usually made of silvered quartz. Other instruments to accomplish a like purpose are made in Cambridge, England, by the Cambridge Scientific Instrument Company, by Wertheim-Solomonsen of Amsterdam, by Lorenz of Berlin, and by Kunsch and Jaeger of Rixdorf. The poles of the magnet are perforated and illuminated, and the galvanometer, suspended at right angles to the perforations, is charged by the current of the magnetic field, while a portion of the shadow of the vibrating wire, magni-

fied by a microscopic lens, is thrown on the moving sensitive plate and photographed.

As the open space is a very narrow slit and the wire is at right angles to it, the shadow thrown on the paper is that of a minute quardrilateral. Now, this little quadrilateral, a mere speck, vibrates back and forth, throwing its shadow on the moving plate, the successive vibrations corresponding to the particular cardiac muscle that contracts. The greatest contraction, which is that of the ventricles, forms the high notch in the tracing; the smaller notch that precedes the bigger one is the contraction of the auricles. The tracing does not distinguish between the right and left

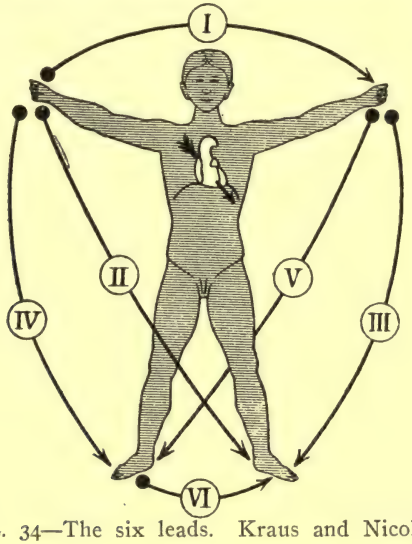

Fig. 34—The six leads. Kraus and Nicolai.

ventricles, or right and left auricles, but records the sum of the contractions of the two ventricles and two auricles. These are the main characteristics of every tracing. There are subordinate notches to be described later.

The electromagnets, constituting the north and south poles, are fed by an accumulator battery of about ten volts, which is a necessary part of the outfit. The principle on which the galvanometer works is that an electric conductor actuated by a current placed in a magnetic field at right angles to the magnetic current oscillates forward and backward, according as the current is ascending or descending, as with the oscillating current of the street supply. The amount of oscillation depends on the strength

of the magnetic field, the strength of the street current, and the resistance.

But besides the accumulator battery, the machine requires a "null" apparatus. For when the hands or arms or feet are immersed in salt water, a so-called "null" or body current develops, and it has to be eliminated or "compensated for" in some way. Accordingly, on the table of the machine there is a "null" aparatus, or "condenser," which must be placed in the line of the electric current.

Another apparatus to be fitted to the table is an appliance for regulating the voltage or amperage. Wheatstone's bridge is also used in addition for estimating the resistance of the electrodes and the body of the patient.

In Fig. 32 is seen an Einthoven galvanometer, *J*, suspended be-

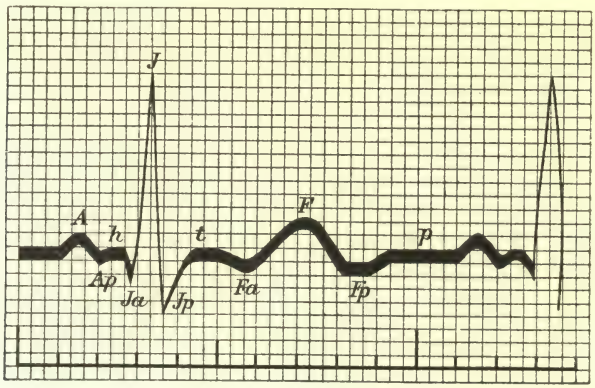

FIG. 35—Schematic representation of a normal electrocardiogram. Einthoven. *A*, Auricular notch or wave. *J*, Initial notch of ventricular contraction, *F*, Final notch of ventricular contraction. *Ap*, Negative notch following auricular notch. *Jp*, Negative notch following initial ventricular notch. *Fp*, Negative notch following final ventricular notch. *Ja*, Negative notch preceding initial ventricular notch. *Fa*, Negative notch preceding final ventricular notch. *h*, Period during which there is activity in His's bundle. *t*, Continued contraction of ventricle. *p*, Period of cardiac inaction.

tween the poles of two electrodes. The electromagnets are wound with copper wire, and set in an iron frame, *W*, supported on adjustable feet, *DD*. A microscope, *R*, pierces the axis of the magnets, and is illuminated by a lighting appliance, *C*. An apochromatic lens is fitted into the tube, *R*, and another into the tube *C*. The first magnifies, and the second casts the shadow on the sensitive plate, operating through a narrow cleft which causes the shadow in the form of a minute right-angled speck to write on

the plate as it vibrates back and forth. It is no easy matter to throw the shadow on the paper. Careful adjustments have to be made by the micrometer screws, *L* and *Q*, so as to throw the shadow into the middle of the field, while the adjusting micrometers, *G* and *F*, have to be turned until all color defects have been eliminated. The plugs, *A A*, are used to shut off light currents of air, etc., that might disturb the electric currents.

Accordingly, for heart work, there is needed to make the apparatus complete: (1) A string galvanometer; (2) a lamp; (3) electrodes; (4) an accumulator battery; (5) an apparatus for testing the susceptibility of the galvanometer; (6) an apparatus for compensating the "null element"; (7) a photo-registering apparatus; (8) an achromatic miscroscope.

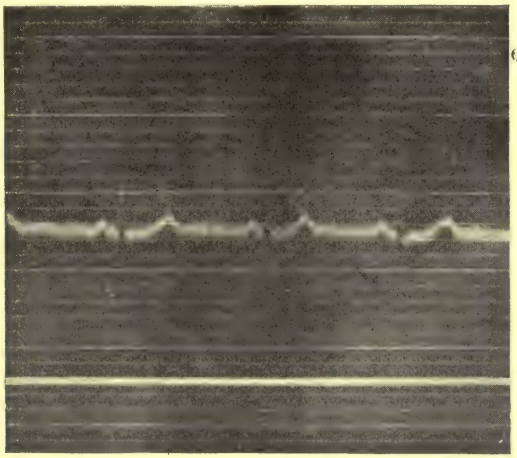

FIG. 36—Electrocardiogram of a normal heart taken by a lead through the right arm and left leg. A 1,000 volt current and a 1 cm. excursion of the needle on the screen were employed. This tracing was taken in the laboratory of Mechanicophysical Institute of Franzensbad, during the summer of 1910.

In Fig. 33 there is seen at *L* an electric arc lamp, the intensity of the flame being regulated by adjusting screws. At *W* is seen the water bath used to absorb the heat rays of the lamp. At *G* is the galvanometer suspended between the poles of the magnets. Alongside of these are the accumulator batteries, while under the lamp and water bath are the "null" apparatus and the apparatus for testing the sensitiveness of the galvanometer.

Now, as a matter of fact, the course of the electric current generated in the muscles of the cardiac chambers is much like that of

the currents that pass over the surface of the heart, as to direction. We know that the left ventricle and right auricle are the heart chambers nearest to the surface of the body, and we also know by animal experimentation that the current through both arms is quite like the current emanating from these two points in the heart. But the current shown at II Fig 34, which passes from the right arm to the left leg, gives the best tracing, as a rule. That is, by referring to the standard of the normal heart, II represents the tracing that is accepted for the normal heart. And it is customary in making any tracing to indicate on

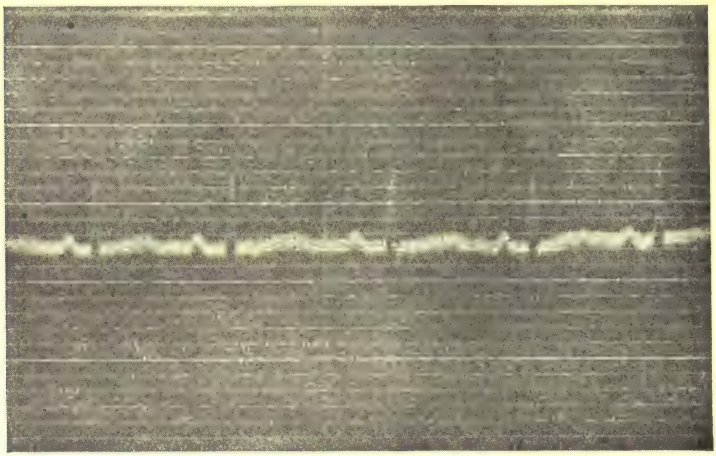

Fig. 37—Electrocardiogram in a case of aortic aneurysm, as shown by the Röentgen ray, with clinical signs of aortic and mitral insufficiency. There is seen to be moderately strong initial contraction of the ventricles, but well pronounced contraction of the auricles. The final contraction of the ventricles is not well marked, but the rhythm is fairly good. (Franzensbad, August 10, 1910.)

it the course of the current, whether as in I, II, III, etc., up to VI. In Fig. 34 the source of the currents is shown; for example, in I, it is both hands, in II, right hand and left foot, in IV, right hand and right foot, and so on. The course of a current is called a "lead."

But of course the strength of the current is modified to some extent by the amount of superlying flesh, and the best records are taken from spare people.

This apparatus should include a couple of hand, arm or foot baths of metal, zinc being generally used, to which the electric wires are attached, while the hands, arms, or feet are immersed

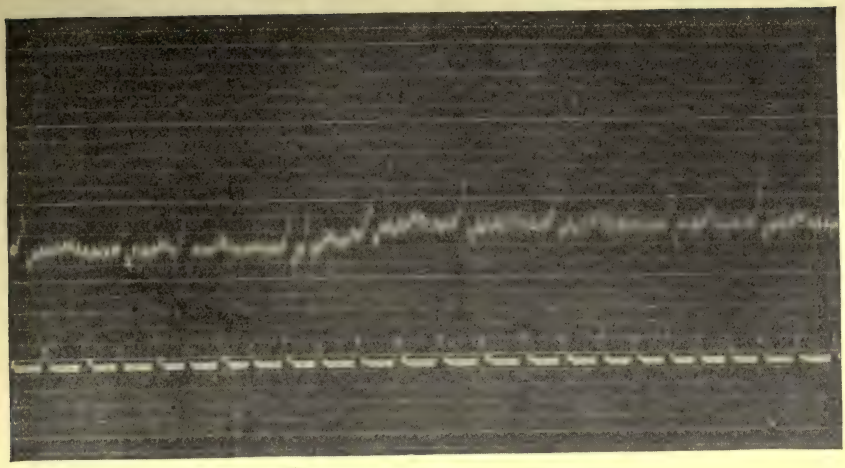

FIG. 38—Myocardial degeneration. Irregularity in action of ventricles and auricles. Extra ventricular systole (at +). Diminished ventricular contraction. Auricular contraction not always defined.

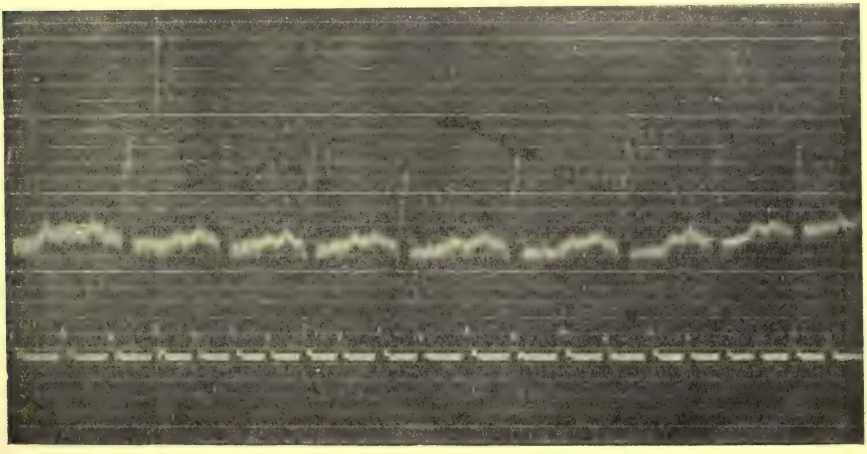

FIG. 39—Mitral insufficiency. Arrhythmia; irregularity in time and force of ventricular contractions; also in time and force of auricular contractions, but the latter are well marked. At + + are extraauricular systoles.

in the salt water of the bath tub. The cost of the whole, as made by Edelmann of Munich, runs from $400 to upward of $1,000, and may be much more, especially if duty is paid on it.

Fig. 35 is a scheme made to represent the electrocardiogram of

a healthy adult, and the notches or waves are indicated by letters. It is best to take the tracing when the patient is in the reclining, not the sitting, position. It may be said here that this graphic tracing of the auricular contraction is apt to be better than if taken from the veins of the neck by any other registering apparatus. Moreover, these notches are apt to be well shown in cases of arteriosclerosis and in heart block.

In Fig. 38, at + is seen an extraventricular systole. The diminished contractile force of the ventricles is shown by the comparative shortness of the ventricular notch. The juvenile type of it is also shown by the fact that it invariably drops beneath the level of the other tracings. On the lower line is the record of the time marker. Each abscissa represents one-third of a second. Owing to the fact that the sensitive paper did not move with uniform speed, the time marking is not absolutely, though nearly uniform. This is one of the minor errors to which such delicate instruments are liable.

The absence of the auricular notch is apt to be ascribed to some affection around the Keith or Tawara node causing disturbance of transmission.

An effort has been made to construct from a large number of electrograms the tracings of certain well-known forms of valvular disease, such as mitral stenosis, aortic insufficiency, and mitral insufficiency. The difficulty is not so great with mitral stenosis, which in its developed form has characteristic clinical features, and would naturally, at least when there is the "check" that is associated with the presystolic murmur, give a characteristic tracing. But these lesions are so generally intermixed, *i.e.*, the valvular lesions are so apt to be multiple, that it must be impossible in the vast majority of cases to dissociate them. Indeed until autopsies have been made in sufficiently large numbers to furnish tracings for individual single lesions we shall not be able to say this or that curve belongs to this or that lesion.

Inasmuch as this presence of a single lesion is so very infrequent, the call for a special curve tracing to diagnosticate a special valve lesion becomes of less importance from a diagnostic point of view.

It is important to control the electrocardiogram by means of other methods of graphic tracing so that one may help to interpret the other. Kraus and Nicolai believe, however, that the electrocardiogram is even now applicable for clinical medicine, al-

though it must be admitted there is still a lack of correspondence between physiological and clinical tracings. To their minds, however, a diagnosis in heart cases that embraces chemical, microscopic, and bacteriological methods is not complete without this instrument. Briefly, they say (1) that the electrocardiogram depicts the precise nature of the heart's contractions more completely than any other method; (2) that the movements of the auricles are specially well shown; (3) that the time and place of the irregularities are also shown, and (4) that it augments the means of diagnosis and may be used to control other methods.

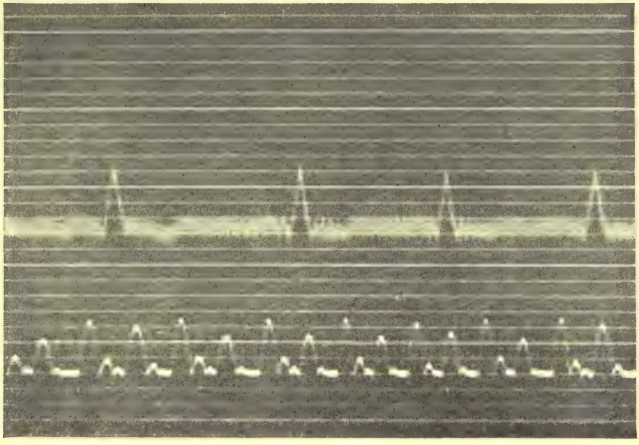

Fig. 40—General arteriosclerosis with anginal attacks. Showing negative phase following initial ventricular contraction in place of positive phase (inverse type). This is regarded by Kraus and Nicolai as an important sign of beginning arteriosclerosis, and is apt to be associated with cardiac insufficiency. (Franzensbad, July 20, 1910.)

And yet, according to Pribram of Prague, there is really no stanlard tracing for healthy adults, though in most instances they tally with Einthoven's curve. Occasionally patients with abnormal conditions of heart and lungs will yield normal tracings.

Sometimes there is a sixth notch. (See Fig. 35.) In children the notches are shorter, and often in persons of powerful build they are higher, but it is not always so. In the pulsus irregularis perpetuus the auricular notch is absent.

According to my notions this method is not, as yet, taking it all in all, so valuable for diagnosis as the polygraphic is or blood pressure or X-ray work; it is rather an accessory to other means of diagnosis. Though my personal experience with it has

been limited, I have seen enough of it to convince me that it has not yet reached the stage where it is worth while for the ordinary practitioner to depend on it in clinical work. Moreover, it is very complicated, consisting of a number of separate apparatuses, requiring much skill to operate. It is also costly, and is very easily put out of condition.

From what has been stated it is apparent that the sensational statements which have appeared in the public journals as to the utility of the instrument must be received with much caution. The electrocardiogram does not as yet of itself distinguish uniformly functional from organic heart diseases. Neither can the use of the instrument dispense with the personal examination of the patient. Hence a complete diagnosis can not be made by the instrument when the patient and the physisian are far apart. Neither the electrographic machine nor any other instrument of so-called precision tells an invariably truthful story. Mechanical difficulties will at times occur, making the picture untruthful. Moreover, as already said, the standard normal curve has not been agreed upon. Strictly speaking, Einthoven has not inaugurated a new method; he has utilized the photographic method of Marey, and the string galvanometer of Ader, while the credit of the completed instrument is due to Edelmann. Einthoven has, however, given close study to the method with the perfected instrument, and he is, therefore, entitled, more than any other man, to have electrocardiography associated with his name.

This much may be truthfully said: The instrument is capable of showing the time and force of action of ventricles and auricles; notes lack of transmission; and demonstrates ventricular and auricular extra-systoles more clearly than any other method. It is certainly useful in various forms of arrythmias, particularly when there is more or less complete auriculo-ventricular dissociation. Electrocardiography has already broadened the knowledge obtained by other graphic methods. But at present the initial cost of the Einthoven machine, and the expense connected with operating and maintaining it, will be a bar to its general use, except in the larger hospitals or research laboratories.

THE SPRING RECORDER

AND A NEW TIME MARKER.

For more than seven years I have been using in my office an automatic lever and spring recording machine, my first experiments with it having been made on March 4, 1904, when I had the assistance of Mr. John T. Hoyt, of the Department of Physiology, Columbia University.

The pen arm of the machine is actuated automatically by the levers and spring of the ordinary bath-room or office scales, on the platform of which the patient stands, sits, or lies horizontally on a raised seat firmly attached to the base. For the patient may sit or stand on the platform of the Chatillon weighing scales[2]; while by elevating the wings of the seat it is converted into a couch on which he extends himself at full length. The action of the heart and blood vessels is communicated to the special pen arm on the dial (Fig. 41, *A*), which is then able to record the cardiovascular waves automatically on the smoked paper of a revolving drum *B*. The name cardiovascular is given to these tracings because they represent the action of heart and vessels conjointly. The pen arm, made of aluminum, is heavily shaded in the diagram. Below it, another pen arm, made of rye straw and tipped with platinum, celluloid, or tin foil, receives the impulse of the carotid or jugular, or liver pulse, or of the apex beat, as may be desired, through a brass receiver *D*, and writes the curve on the same paper. Still below this, the metal pen arm of a Jaquet chronograph, *C*, or an electric time marker, may register the time simultaneously on the drum in seconds or fractions of seconds.

In Fig. 42 is seen a cardiovascular tracing taken by the recorder, showing a pulse of irregular force and rhythm. It also gives the respiratory curve.

In Fig. 43 is seen another cardiovascular tracing taken by the same instrument, but three times larger than the original. An enlarged tracing is always obtainable where there is considerable

[2] John Chatillion and Sons, 85 Cliff street, New York.

cardiovascular force, delicate sensitiveness of the scales, and considerable speed of the drum.

If we accept Einthoven's explanation of the significance of the waves of the electrocardiogram, as given below (Fig. 44), the interpretation of these tracings is as follows: The tall or verti-

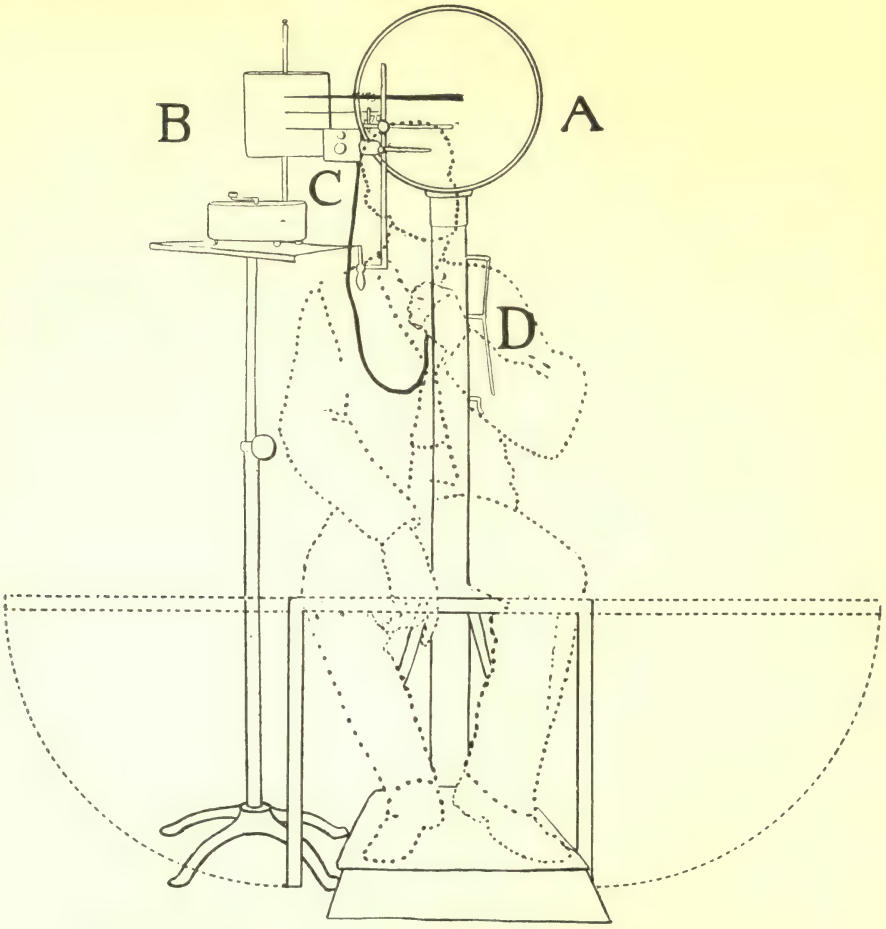

Fig. 41—Automatic lever and spring polygraphic machine or recorder.

cal stroke indicates the ventricular systole, the second also representing the ventricular contraction, or the tidal wave; the third wave the end of ventricular contraction, or the dicrotic wave. The fourth and fifth waves represent auricular contraction. The sixth wave, if it exists, indicates the activity of His's muscle bundle.

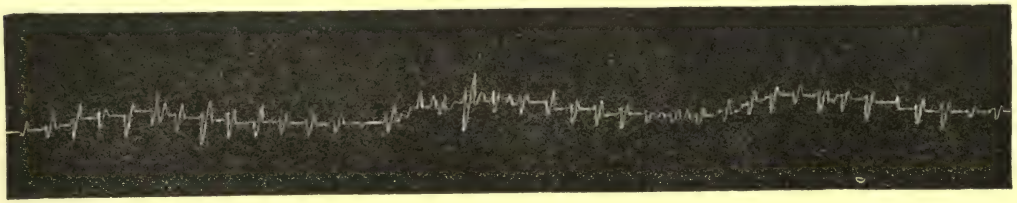

FIG. 42—Cardiovascular tracing, showing an irregular respiratory curve, in a pulse irregular as to force and rhythm.

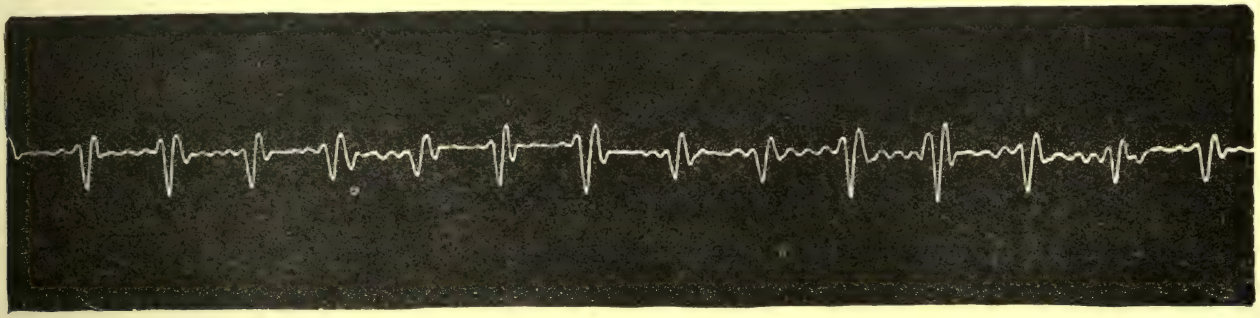

FIG. 43—Cardiovascular waves as taken by the automatic recorder. From the original, magnified three times.

It will be noted that there is a fair degree of coincidence between the general characteristics of the tracings made by the two machines.

While the automatic lever and spring instrument is capable of doing the work of an ordinary polygraphic machine, aided by the usual receivers, tubes, tambours, and pen arms of polygraphic machines, I have used it more especially in studying the effects of nicotine, spirit of nitroglycerin, and caffein on the human subject, in respect to the rapidity, duration, and force of their action on the circulatory system. In these experiments, polygrams are able to furnish with reasonable accuracy a graphic record of the rate and rhythm of pulse and respiration, and of their comparative force, before, during, and after the several experiments. The requisite measurements for the determination of these several items are taken with calipers and a millimeter rule. In this way also a comparison can be instituted between control experiments and the direct ones. In respect to the testing of drugs this recorder represents a new kind of instrument, particular applicable to the study of pharmacodynamics. So far as I know, this and the electrocardiographic machines are the only instruments that

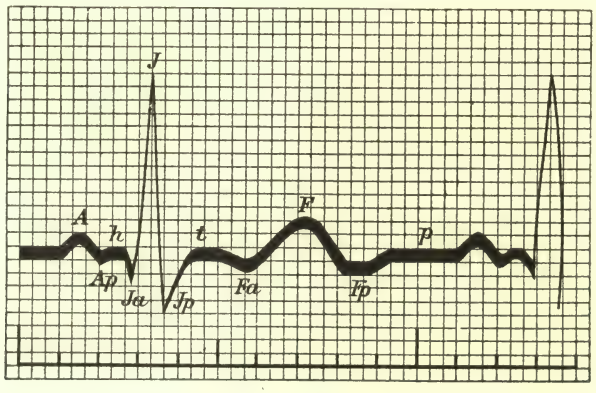

FIG. 44—Einthoven's schematic representation of the successive events in the cardiac cycle of an electrocardiogram. *A*, auricular wave; *h*, small wave indicating activity of His's muscle bundle; *J*, large initial wave of ventricular contraction; *t*, secondary contraction of ventricle; *F*, final larger wave of ventricular contraction; *p*, period of cardiac inaction, preceded by an imperfect wave. *Ap, Ja, Jp, Fa, Fp,* negative waves following muscle contractions.

The rectangular rulings of this diagram enable the reader to estimate Einthoven's conception of the comparative dimensions of the several events in a cardiac cycle.

register automatically the successive events in a cardiac cycle. The distinctness of the respiratory curve, as seen in Fig. 42, is also a noteworthy feature of this automatic recorder.

A NEW ELECTRIC TIME MARKER.

Owing to the length of time required to procure a Jaquet Time Marker, its initial expense, duties, and express charges, I have undertaken to get up an American Time Marker, which can be made at home, and which involves no great expense.

In the accompanying cut (Fig. 45) is seen my instrument, whose motive power is actuated by two ordinary dry cell batteries, *AA*. These are connected by wires with the two more distant binding posts, *dd*, of the switch-board, *D*, the current actuating the platinum interrupter, *a*, of the clock, *H*, and also the wires connected with the two nearer binding posts, *dd*, by the

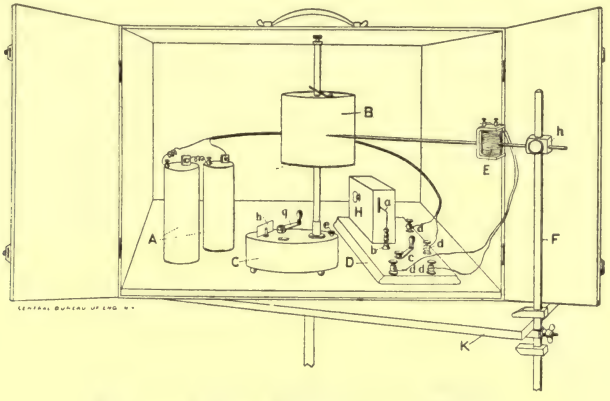

FIG. 45—A New Electric Time Marker

A, Dry cell batteries. *B*, Aluminum drum. *C*, Slow and fast Kymograph clock, with adjustable bearings. *D*, Switch-board. *E*, Signal magnet with pen-arm. *F*, Tangent stand. *H*, Clock for electric time-marker. *K*, Shelf of metal table. *a*, Vibrating interrupter. *b*, Mercury cup. *c*, Switch. *dd*, Binding posts. *e*, Milled head for altering speed. *h*, Fan for regulating speed. *q*, Clock key.

This entire apparatus may be easily contained in a wooden box 9x18x18 inches, as seen in the above cut.

switch, *c*. The current passes ultimately to the signal magnet and vibrator *E*. The pen-arm or lever of this magnet vibrates as often as 100 times per second. Fine adjustment screws regulate the excursions of the pen-arm.

The kymograph used is the one known as the Slow and Fast Combination model of the Harvard Apparatus Company of Boston.[3] The aluminum drum can be moved up and down on an up-

[3] P. O. address, Back Bay P. O., Boston, Mass.

right post, and set at any desired height by means of a spring. By raising or depressing the milled head, *e*, the speed of the drum can be altered, while by the fan, *h*, it can be further regulated. By adjustable bearings underneath, *C*, the apparatus can be leveled. The tangent stand, *F*, is attached to the metal shelf, *K*, which can be raised or lowered to the required height. The

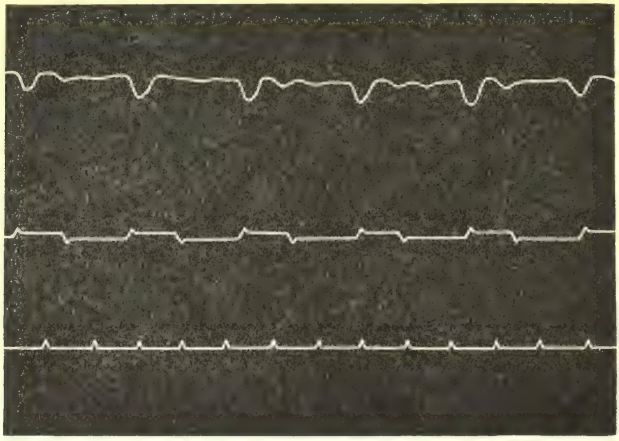

FIG. 46—Cardiovascular tracing taken by the author with his Automatic Spring Recorder. Simultaneous tracings of the time abscissae in 1/5 seconds by the Jaquet instrument (lower tracing), and the author's Electric Time Marker (upper tracing) in abscissae of 2/10 and 3/10 seconds. Rate of pulse, 75.

wooden box containing the appliances when placed on the metal shelf, can be turned about in any desired position, while the kymograph and switch-board can also be moved about. Kymographic paper may be obtained from the Harvard Apparatus

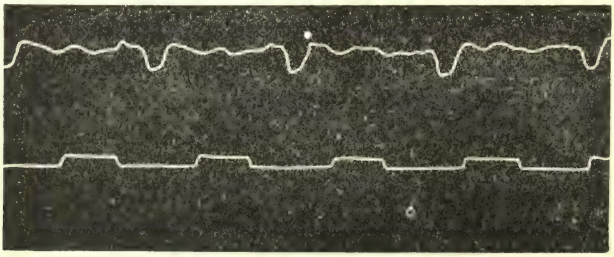

FIG. 47—The upper tracing, taken by the author's Automatic Recorder, shows fairly well the five waves of the Einthoven electrogram, including the final wave of the cycle, attributed to the action of the His bundle. The lower tracing is that of the author's Electric Time Marker. Small abscissae 4/10 seconds, large 6/10. Rate of pulse, 60.

Company. My switch-board, clock, interrupter, pen-arms, and electric connections, were furnished by an electrician.[4]

It is evident that graphic methods are useful not only in diagnosis with reference to treatment, but also as a register of actual facts. Certainly no large institution should be without a good polygraph.

[4] E. J. Dustman, 167 East 72nd Street, New York City.

Chapter V.

SOME OF THE NEWER INSTRUMENTS OF PRECISION.

During the past few years a number of ingenious appliances have been devised as aids in medical diagnosis. Some of them have been improvements on previous models; others are entirely new instruments. The Hirschfelder modification of the Erlanger sphygmomanometer is an example of the one kind, and the micrograph of the other. But none of these instruments have been known to practising physicians in the degree that their importance has warranted; in fact, in most instances they have been hidden away in foreign or special journals, or merely figured in the catalogues of instrument makers. This being the case, I have undertaken to furnish a description of some of the more important, knowing that our medical public is always appreciative of any appliance that can throw additional light on the many problems of diagnosis that constantly confront us in practice. I shall describe briefly the Hirschfelder modification of the Erlanger sphygmomanometer, the Uskoff sphygmotonograph, the phonoscope, the micrograph, and the viscosimeter.

THE ERLANGER SPHYGMOMANOMETER AND HIRSCHFELDER POLYGRAPH

The Erlanger sphygmomanometer has recently been made more elaborate by having attached to it the Hirschfelder polygraphic machine (Fig. 48). It combines sphygmomanometry and polygraphy. The patient lies in a nearly horizontal position, with the head and neck supported by a single pillow. The cuff is adjusted for the right arm, and the maximal and minimal blood pressure is taken. Then two additional levers are mounted on a tangent stand, and connected with long pieces of rubber tubing attached to the Mackenzie receivers for the jugular and apex or carotid. When the jugular is to be taken, the patient turns his head to the right and downward, so as to relax the right sterno-mastoid. To take the brachial tracing, the cuff is inflated at minimal pressure. The carotid pulse is taken by putting a small tambour over the carotid when it is beating actively. In this way three separate tracings may be obtained. The interpretation is the same as given by Mackenzie.

THE USKOFF SPHYGMOTONOGRAPH.

The Uskoff instrument, known as the sphygmotonograph, (Fig. 49) records simultaneously in millimeters of mercury the blood pres-

sure of the carotid or jugular pulse, the apex beat, the brachial, and the time in fifths of seconds.

The method of using this instrument is as follows:

The cuff, *M*, is attached in the usual manner to the upper arm and pressure applied by means of the bulb, *B*, until the pulse disappears. The pressure thus applied is first transmitted to the mercury manometer, *Q*, lifting the perforated float as the mercury column rises. At the same time it is transmitted to the rubber

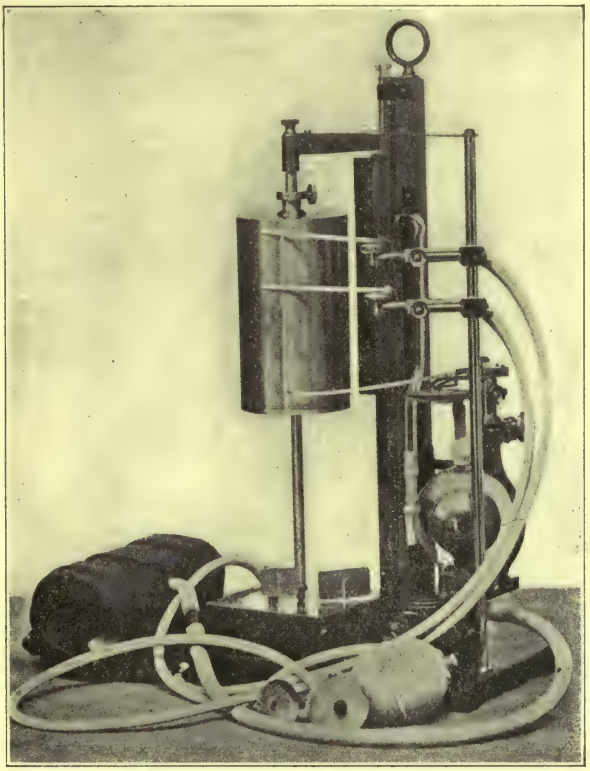

FIG. 48—Erlanger Sphygmomanometer with Hirschfelder Polygraph.

bulb enclosed in the glass bulb, *G*, which is in air-tight connection with both the cuff and the manometer. With the handle of the cock, H_1, in the vertical position, the air in the glass bulb is expelled through the cock, H_2, which should be opened at the same time as H_1.

When the pulse has, by application of sufficient pressure, disappeared, the cock, H_1, is turned in the direction of the arrow,

which movement connects the glass bulb, *G*, with the outside air through an exceedingly small opening and the pulsations appear because air pressure in the cuff and manometer is thus slowly relieved through the valve, *V*, and is divided between the rubber bulb and the air in the glass bulb, *G*. The pulsations are at the same time transmitted from the air in the glass bulb, *G*, by means of the third tubulature in connection with the indicator, to the tracing paper.

The pressure arising from compression of the rubber bulb, *B*, is also transmitted to two small outlets confronting each other on the top of the manometer and between which openings the perforated float passes as it rises and falls on the mercury column. The

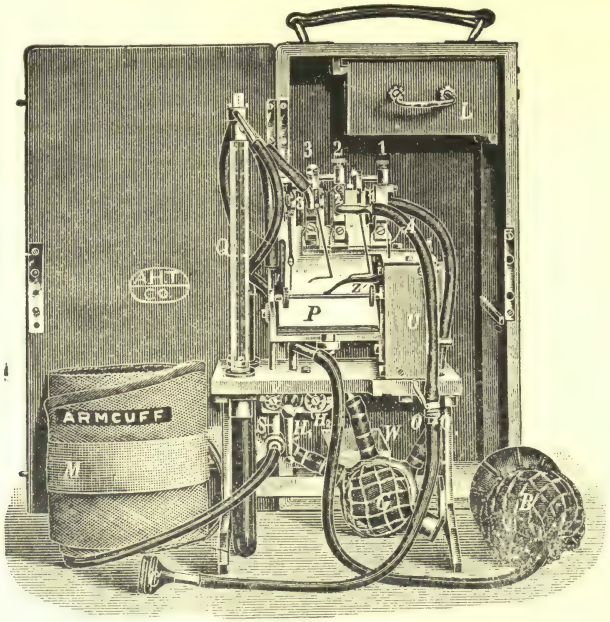

Fig. 49—Uskoff Sphygmotonograph.

small holes in the float are arranged 1 mm. apart with outstanding marks, indicating 50 millimeters of mercury. The passage of the air current through the small openings and through the corresponding perforations in the float, operates indicator 3, and thus graphically records the actual pressure in millimeters of mercury at any moment during the operation.

In Fig. 50 the upper tracing, *A*, represents the blood pressure in millimeters of mercury, and the tracing is made while the

pressure is reduced from 200 mm. to 50 mm., each vibration representing 2 mm. of the mercury column.

The second tracing, *B*, represents one taken from the carotid artery and recorded by indicator 2. This indicator may be used to record either the apex beat, the jugular or other vein pulsations at will.

The third tracing, *C*, is the sphygmotonogram or the tracing of the arterial (brachial) pulse under a falling pressure. With 200 mm. of pressure this indicator traces a straight line because of the total obliteration of the pulse. At 162 mm. of pressure the pulse reappears and at 92 mm. of pressure has reached the lowest point of maximum oscillation. If the float on the mercury column continues to sink, the internal pressure is shown to be greater and the vibrations become smaller and smaller because of the elastic layer between the cuff and the artery, until they disappear entirely. This

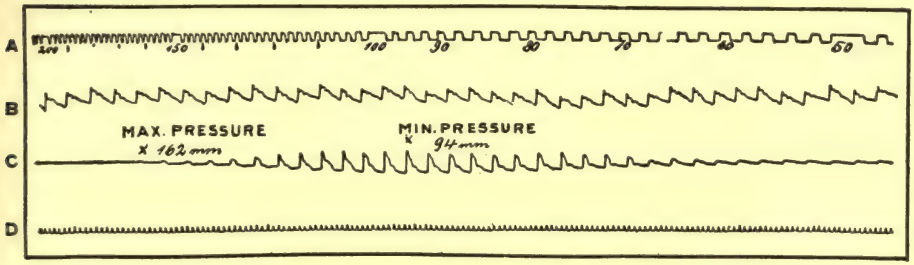

Fig. 50—Specimen Tracings taken by the Sphygmotonograph.

tracing taken under diminishing pressure constitutes, therefore, an accurate record of both the diastolic and systolic pressure.

Arthur H. Thomas of Philadelphia is the agent for this instrument.

PHONOCARDIOGRAPHY.

A special method of registering heart sounds and murmurs has been devised by Professors Joachim and Weiss of Königsberg, in Prussia. It was first described in 1908.[1] The sounds and murmurs are registered by photography, and are then reproduced. Recently these experimenters have revised their work, publishing their results as applicable to diagnosis in valvular affections.

The sensitive membrane which receives the sounds is composed of a mixture of soapy water and gelatine, prepared as follows: To one liter of warm distilled water is added 25 grams of Marseilles

[1] Joachim und Weiss, *Arch. f. Phys.*, 1908, CXXIII. 341; *Deutsch. Arch. f. Klin. Med.*, CLXXXVIII, xcviii. 513.

soap. After thorough solution it is cooled, and 660 grams of glycerine added. This is again cooled down and filtered until the solution is quite clear. It can then be kept indefinitely without change, in closed vessels. This preparation is used as a sensitive membrane to receive the sounds in the phonoscope, and it is claimed that this saponaceous membrane surpasses the most delicate microphone in sensitive qualities, as a receiver.

As a medium for conveying the oscillations of the membrane there is a silvered glass filament, bent at an angle, planted in the center of the membrane. The oscillations of the rod are photographed by a registering photographic apparatus, actuated by clockwork, and similar to the machine used in the Einthoven cardiographic machine. The membrane and glass lever are fitted into a case called a phonoscope. This is a small metal box resting on a vertical support, provided with three adjustable feet. In one side is a glass window, having a diameter of about one and a quarter inches. On another wall there is a shutter, in the middle of which is a tube, into which a socket provided with a circular opening over which the soapy membrane is drawn. The glass lever is attached to the top of the box by shellac, while the opposite end, forming a loop, is planted in the soapy membrane. The sliding shutter enables the operator to move the lever into the required position.

The glass filament is illuminated by an objective in the tube, while another objective projects the image of the filament on the sensitive paper. A telephone sirene is then introduced in a telephone circuit, and the ear hears the sound in the telephone receiver.

This is an improvement on the apparatuses of Huerthle,[2] Donders, and others. Huerthle's method was to place a stethoscope over the site of the apex beat; on the opposite end of this instrument was a resonator made of a hollow wooden cone, in the apex of which was screwed a wooden rod. This latter was the carrier of twenty-four thin plates of wood to increase the resonance, while on the end of the wooden rod was a wooden tuning fork. Between the branches of this was a microphone, the contact being made by means of opposing armatures of silver and carbon. By connecting this instrument with the telephone the heart sounds were conveyed to the listener.

THE MICROGRAPH.

The micrograph is an instrument that magnifies the vibrations of a diaphragm, similar to that of the telephone, about 50,000 times, and records them on a moving photographic film. The word micro-

graph is used to indicate that it records microscopic movements. It was devised by Albert C. Crehore, Ph.D.[2] The apparatus consists of the instrument proper, and the photographic attachments. An ordinary receiver, connected with a flexible rubber tube, conveys the vibrations through a column of air to the diaphragm, attached to which and parallel to it is a cylindrical lens, whose plane surface is turned to the reflector.

Now, if a source of light (in this case the mercury-vapor lamp of the Cooper-Hewitt system) is thrown on the surfaces of the reflector and lens, the observer will see a series of interference bands on the surface of the reflector. These comprise a number of concentric circles, which by means of another lens can be thrown on the ground glass of a camera.

If, now, a slit is made to intervene between the lens just mentioned and the photographic plate, so as to cut off all the light of the image except that which passes through it to the plate, and the plate is moved across the slit at a uniform rate, the several bright areas of light along the diameter of the image will trace lines on the plate, and the developed negative will show a corresponding series of wavy bands. These bands are constantly changing, according as the lens and reflector approach or recede from each other, as they do during the vibrations caused by the movements in the closed currents of air. The well-known principle of light interference, discovered by Young in 1801, is used to produce the waves. As the photographic film is carried along by a motor at a given rate of speed, the length of the waves and the wavelets in the cardiac cycle is easily determined. By means of a pair of dividers to measure the time intervals from band to band, and by counting the number of bands, a curve can be plotted with accuracy.

By means of this instrument, not only can tracings be taken of the apex beat, the radial and the jugular, but heart sounds can also be registered. By using the ordinary armlet, systolic and diastolic pressures can also be photographed, and determined subsequently by a scale furnished for the purpose. In fact, this ingenious instrument is apparently destined to aid us materially, in conjunction with the electrocardiograph and the polygraph, in further elucidating the problems of the cardiovascular mechanism.

THE VISCOSIMETER.

During the past ten years, a good deal of attention has been given to estimating the viscosity of the blood, especially with refer-

[2] *Arch. f. Phys.*, 1895, LX. 263.

[3] Through the courtesy of Dr. Frank S. Meara I have seen it in operation.

ence to its bearings on clinical medicine and surgery. The method of determining viscosity is not difficult, requiring only a simple and inexpensive instrument. All that is necessary is to note the time taken by a given quantity of blood in passing through a glass tube of definite calibre and length, and then to compare this period with the period required for distilled water to pass through another tube of similar bore and length. The index of viscosity is expressed in numerals, such as 1.50, 2, 4, 5, etc., which denote that the time occupied in passing through the tube is one and a half, two, four, or five times that of distilled water, the standard being 1. The normal viscosity is set at from 4.25 to 5.

The instrument itself is called a viscosimeter, and the prinicpal models, called after those who have designed them, are the Huerthle, Hirsch and Beck, A. Mayer, Demning and Watson, Tissot, Burton-Opitz, Determann, Hess, Muenzer and Bloch, MacCaskey, Zangger and Scarpa. These instruments vary as to reliability, but in the best the error is said to be not more than 2 per cent, a negligible quantity.

At first it was thought that viscosity bore a tolerably uniform ratio to the hemoglobin content, the corpusle count and the specific gravity of the blood. But so far as leukemia at least is concerned, with a viscosity of 5.12 or slightly above the normal bound, there has been found to be a hemoglobin content of 60, the normal being about 100. Determann[4] has also found that it is not the corpuscle count that preserves a fairly uniform ratio with the viscosity, but rather the volume of the combined red and white corpuscles; while it has also been found that from 4 to 20 per cent. more of viscosity resides in the white corpuscles than in the red. The material causing viscosity is in their interior, and is liberated when they disintegrate. The specific weight of the blood has been found to have nothing to do with the viscosity.

On the other hand, the gaseous content of the blood has much to do with it. When this fluid becomes oxygenated in the lungs, it furnishes oxygen to the tissues, in return for carbonic acid gas, while the chlor-ions are absorbed by the corpuscles, together with water from the serum, and thereby the corpuscles become swollen. This increase in viscosity is shown in a comparison between venous and arterial blood. But serum-albumin also increases viscosity, though not to the extent of the sodium salt. According to Adam,[5] viscosity is reduced by the iodides and bromides of potassium; in fact, other drugs affect it.

[4] Determann: Die Viscosität des Menschliches Blutes, Wiesbaden, 1910.
[5] *Zeitschrift f. Klin. Med.*, 1909, LXVIII. 177.

Viscosity increases also after severe physical exercise, and is modified by diet. So far as the increase in viscosity in cardiac and pulmonary affections is concerned, Determann thinks that it does not affect the circulation uniformly. It is compensated for in some way. The use of the cold bath increases the viscosity; the hot bath reduces it at first, but raises it subsequently to the normal or above it.

From a series of researches by Oehlecker[6] in acute surgical diseases of the abdomen, classed as simple appendicitis, appendicitis with localized peritonitis, and diffuse purulent peritonitis, it was found that in the first class the viscosity was 4.5; *i.e.*, it was within the normal range. In the second class it averaged 5.3, or was raised slightly above the normal; while in diffuse peritonitis it varied from 5 to 6.3, and in two fatal cases rose to 7 and 7.1. Judging from these facts, it appears that the viscosimeter may help to determine the extent of peritoneal involvement. And inasmuch as hemorrhage is associated with a viscosity that is low proportionately to the amount of blood lost, the estimate of viscosity in such cases may help in the differential diagnosis between simple appendicitis, adnexitis, ruptured ectopic pregnancy, or, in fact, any form of internal hemorrhage. Sex, age, and constitution, however, apparently have an influence in increasing or decreasing viscosity.

It will be seen, therefore, even with our fragmentary knowledge of the relation of viscosity to morbid states, that there is a promise of valuable results from the use of the viscosimeter. The subject is certainly one of interest, but requires more extended observation than has been given it. The estimation of viscosity may eventually be of practical use to practitioners—in fact, it is quite within the bounds of possibility that further researches will alter our misty ideas of its value as a diagnostic resource—but at present it is of more interest to the physiologist than to the practitioner, and we are not yet justified in recommending the viscosimeter as an important addition to the present armamentarium of the physician's office, or of the hospital.

According to Burton-Opitz,[7] one of the most recent writers on the subject, the rapidity of the blood current depends on three factors: The driving force, the peripheral resistance, and the viscosity, of which the latter is the feeblest factor. Naturally, the rapidity of the flow varies inversely with the viscosity; *i.e.*, the

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[6] *Berl. Klin. Woch.*, 1910, XLVII. 578.

[7] *Jour. of the Amer. Med. Assoc.*, 1911, LVII. 353.

greater the viscosity, the more is the current of blood slowed. Under ordinary circumstances, however, increased action of the heart or dilatation of vessels overcomes the viscosity; but if this action, which is automatic, is inhibited by disease or otherwise, the slowing effect of the viscosity will not be overcome, though it may be decreased artificially by the use of moderately large quantities of normal saline solution. But Opitz believes that clinicians have attached too much importance to the matter.

Chapter VI.

CARDIAC ARRHYTHMIAS.

The subject of cardiac arrhythmias took on a new phase as soon as graphic methods were used for their differentiation. Indeed, the use of graphic instruments has revolutionized the matter by disclosing new data of great practical value. Most of these discoveries have been the work of the last ten years. As a single illustration of the lines of investigation and their clinical results, Friberger (Upsala Laek. Foreeand., *Journal Am. Med. Assoc.*, 1912, LVIII. 828), recently undertook to examine the hearts of 321 unselected children between the ages of five and fourteen, taking graphic records of each child. Only about 37 per cent. had regular pulses. Of the remainder about 12 per cent. had great irregularity and about 50 per cent. a moderate amount. The variety of irregularity was what Mackenzie has called "the youthful type," and was about equally common in the two sexes. The etiology was not clearly evident, but Friberger found that in advanced tuberculosis there was arrhythmia in about half of those examined. This is only one of the many fields in which graphic methods are helping the practising physician.

It has now been generally accepted that arrhythmias may be satisfactorily classified with reference to the five physiological attributes of heart muscle, demonstrated by Gaskell in 1882. These are, as is now well known, 1, Rhythmicity, *i.e.*, the faculty of rhythmically initiating a stimulus; 2, Irritability or Excitability, *i.e.*, the capacity for receiving a stimulus; 3, Contractility, *i.e.*, the faculty for responding to a stimulus; 4, Conductivity, *i.e.*, the ability to convey a stimulus; and 5, Tonicity, *i.e.*, the power to maintain cardiac tone.

Corresponding to the first four faculties or attributes, there are four distinct varieties of arrhythmias capable of being demonstrated by graphic tracings.

To the first of the forms to be described I have given the name Pneumogastric Arrhythmia, because of its close relation to pneumogastric influences. It was described by Kussmaul years ago, and has been called vagus, fundamental, sinus, normal, or respiratory arrhythmia. The word sinus implies that it is a variety of the rhythm that originates in the sinus venosus; *i.e.*, the normal rhythm. This pneumogastric arrhythmia is a variation from the standard

cycle within physiological bounds. Though the cardiac cycle varies in length, the systolic phase is little altered comparatively, while the diastolic is considerably lengthened, and this latter feature is the chief characteristic.

In Fig. 51 it is seen in the tracing of the radial pulse immediately after the apneic period of the Cheyne-Stokes respiration of uremia. Kussmaul called it the Pulsus Paradoxus. It is the pulse following a deep inspiration, hence the term respiratory. It can also be caused by the act of swallowing, which is largely regulated by the vagus or pneumogastric; hence the term pneumogastric arrhythmia. In Fig. 1, after the apneic period when respiration was resumed the radial pulse took on this peculiar form of arrhythmia, gradually, however, returning to the former speed and regularity. Apart from graphic tracings, this form of arrhythmia is recognized by the finger on the pulse. It can usually be inhibited by a single dose of atropin (grain 1-60).

It is not well to attribute too much importance to an arrhythmia which is usually quite transient, functional in character, coming and going from slight causes. But exceptionally it may be of a more serious nature, as in tuberculosis, or after infective diseases, especially the eruptive fevers of children. It sometimes occurs in neurasthenia; also in over-dosing by digitalis, and probably other drugs which affect the cardiovascular mechanism. From whatever cause, it is a loss of the normal rhythm, the stimulus to which we believe originates, as I have said, in the sinus, venosus.

In extra-systolic arrhythmia there are extra—that is, as it were, supernumerary—contractions, from stimuli that do not originate in the sinus, though in the main the regular or sinus rhythm is maintained. They are of two principal types; the ventricular, where the contraction originates in the ventricle, and the auricular, where it originates in the auricle. Take an example of what appears to happen in these cases. If for any cause the left ventricle fails to empty itself, the residual blood acting as a stimulus can make the ventricle put in an extra beat before the normal auricular stimulus has passed down to it. On the other hand, in dilatation of the auricle, as for example in advanced mitral stenosis, the incomplete expulsion of the blood into the ventricle may operate to produce a supplementary contraction, which would then be an auricular extra-systole. Strain of auricle or ventricle also may and probably does, produce extra-systoles. They have been called "dwarf" systoles, from their small size; "premature," because the contraction is in advance of the normal period for the contraction; "interpolated," because they

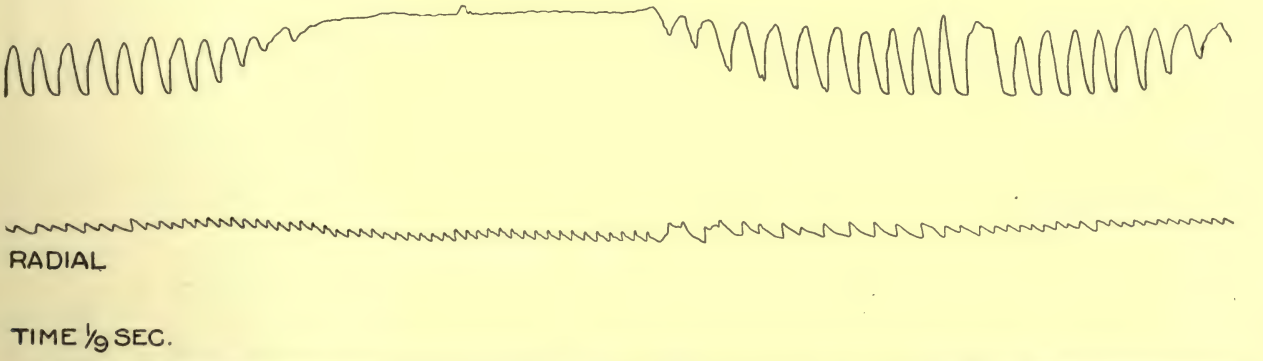

FIG. 51—Pneumogastric, Vagus, Sinus, Respiratory or Fundamental Arrhythmia. Polygraphic tracing taken for the writer by Dr. T. B. Barringer, Jr., March 27, 1909, at Bellevue Hospital, from a uremic patient with Cheyne-Stokes respiration, in the service of Dr. W. Gilman Thompson. During the apneic period shown in the upper tracing (respiratory), the radial pulse became more regular and frequent. In the respiratory phase following the apneic period, the pulse at first was less frequent and quite irregular, but it gradually assumed its former speed and regularity. This is an example of the respiratory type of arrhythmia, and is caused by pneumogastric influence.

are actually interposed between beats that are of the prevailing type at the time.

All extra-systoles are followed by a pulse period that is rather longer than the normal. In the ventricular form the length of the preceding pulse period added to that of the following pulse period is equal to the length of two normal pulse periods. In the auricular form the pulse period following the extra-systole is shorter than the corresponding compensatory pause in the ventricular form.

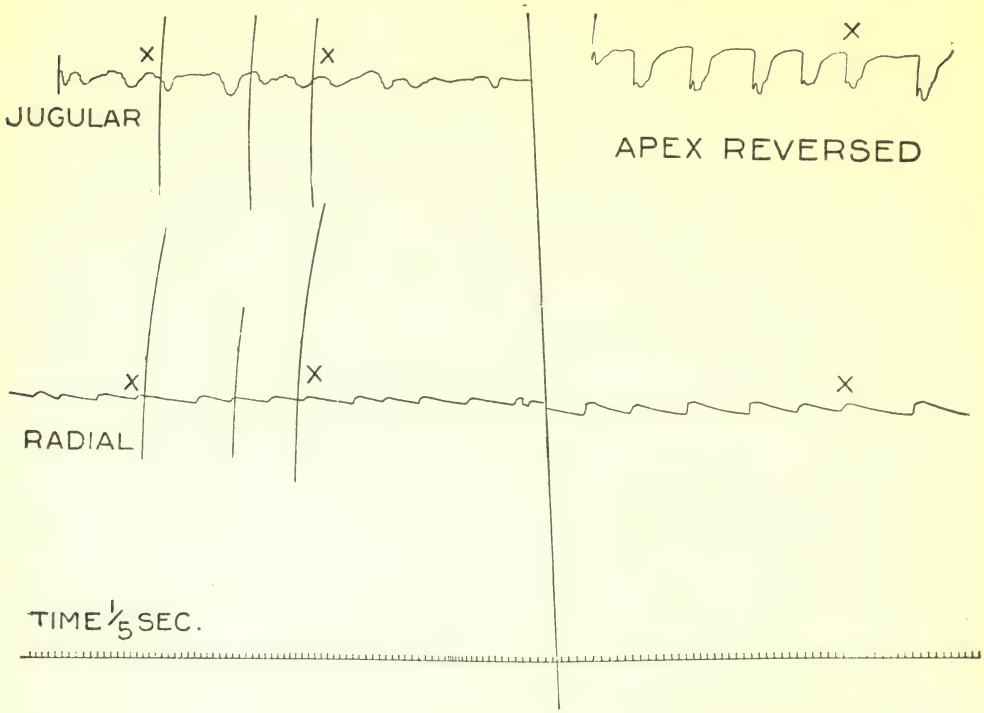

Fig. 52—Extra-Systolic Arrhythmia of the Ventricular Type. Polygraphic tracings taken by Dr. T. B. Barringer, Jr., from a patient with mitral stenosis, tricuspid regurgitation and auricular paralysis. At three points in the radial pulse (XXX) the irregular cardiac rhythm is interrupted by extra-systoles. Inasmuch as the duration of the pulse period preceding and that following the extra-systole is equal to two normal pulse periods, the extra-systole originates probably in the ventricle. The apex beat gives a reverse tracing, because formed by the right ventricle.

Such systoles occur at regular or irregular intervals. They are illustrations of an abnormal irritability or excitability of the heart, and are most common in persons of a neurotic constitution. Sometimes these extra-systoles cannot be detected by the finger, but they are usually recognized in auscultation, when the regular sequence

of beats is occasionally interrupted by one or two short sounds followed by a brief pause.

In Fig. 52, extra-systoles are seen at *XX* in the radiogram, phlebogram and cardiogram. In the radiogram it will be noted that the length of the pulse period preceding the extra-systole, together with the pulse period following, constitute a length of two ordinary pulse periods, a criterion taken to mean that the extra-systole is ventricular, and, according to Mackenzie, that the stimulus originated in the His bundle, on the distal side of the node. But the points of origin of the stimuli may be in other parts of the tissue intermediate between auricle and ventricle. These matters are still engaging the attention of physiologists, and have not been absolutely determined.

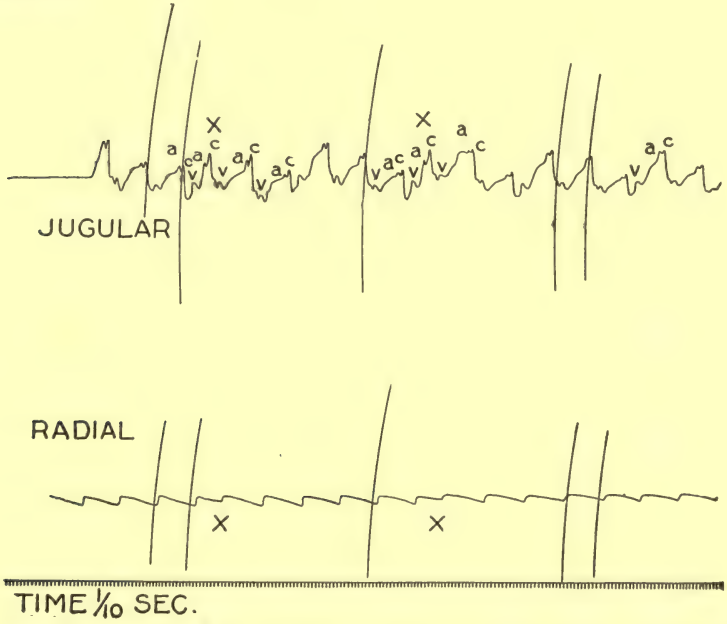

Fig. 53—Extra-Systolic Arrhythmia of the Auricular Type. This polygram, taken by Dr. T. B. Barringer, Jr., represents an extra-systole originating in an auricle. In the jugular tracing the extra-systoles are shown at *XX*. The *a* (auricular) waves represent the contractions of the right auricle. The extra-systoles are shown also in the radial tracing at *XX*. The compensatory pauses in the radial tracing are seen to be shorter than with ventricular extra-systoles.

In the auricular extra-systole the stimulation is thought to arise in the primitive tissue of the auricle. In Fig. 3 these extra-systoles are seen at *XX* in the jugular tracing. A characteristic of this auricular extra-systole is that in the arteriogram the compensatory pause following is shorter than in the ventricular extra-systole, as I have already said.

Extra-systoles occur under the most varying conditions. Coffee, tea, tobacco, and gastro-intestinal distention are examples of determining causes, when their occurrence may not be of very serious importance, though when happening in toxemia, in convalescence, or in weak heart, their import adds to the gravity of the situation. And they may occur together with other cardiac manifestations, when they are a further complication. Not infrequently the extra-systole is appreciable by the patient, as when he feels a sudden thud in the precordium, perhapse with a sense of faintness. It may even seem as if the heart were going to stop; and, as a matter of fact, it may actually do so. I have had such a case, where the heart actually stopped for several seconds; how many I do not know. It may be remembered that a man named Nordini, an Austrian Pole, is said to have the power of stopping his heart for twenty seconds, and his statement has not, I think, been contradicted, as already noted in a previous chapter. In the case of a patient of Dr. Joseph A. Thompson of Philadelphia, a man by the name of Worthington overcome by the fumes of nitric acid was revived by the use of the "pulmotor" after his heart had ceased to act for four hours (New York Press, Sept. 27, 1912). But the extra-systoles are not always appreciable subjectively. To make them distinct, let the patient run around the room a few times and then hold his breath. The extra-systoles are intensified by hurried movements.

The really most important function of the heart muscle is contractility. A striking example of its abnormal characteristics is seen in the alternating pulse (Fig. 54), which consists of an alternation of large and small beats. And the alternation is continuous, which distinguishes this type of pulse from extra-systolic arrhythmias. It may always be recognized and differentiated by graphic methods, when other physical methods leave doubt as to the diagnosis. It is, however, a rarity. I am indebted for the illustration to the courtesy of Dr. George Bachmann of Atlanta, Ga.

Another example of abnormal contractility is seen in auricular fibrillation, formerly called nodal rhythm by Mackenzie, and one cause of the permanently irregular pulse of Hering. Here the cardiac cycles vary so much that there is no sequence of beats having the same length. This special characteristic is well shown in one of my cases of heart block (Fig. 55).

The source of the difficulty is put at the auriculoventricular node, which governs auricular and ventricular contractions through the bundle of His.

In 1905 Cushing and Edwards suggested that in some of these patients the cause might be auricular fibrillation, a condition in which component parts of the muscle wall of the auricle contract independently of one another, and in such a disorderly fashion that it might almost be said auricular contraction as a whole was at a standstill. But it was not until 1909 that researches by Lewis on the lower animals showed, by means of comparison between the arterial and venous pulse tracings and electrocardiograms, that this so-called nodal rhythm, or permanently irregular pulse, was to be attributed to auricular fibrillation.

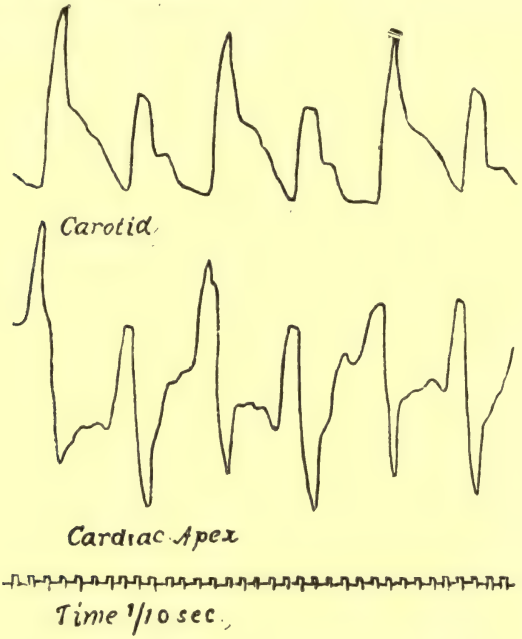

FIG. 54—The Alternating Pulse. From a woman aged 33, with mitral stenosis, aortic regurgitation, and failing compensation. Systolic blood pressure 220 mm. Hg.; diastolic 160 mm. Hg. By Stanton instrument with 12 cm. cuff.
By courtesy of Dr. George Bachmann.

For in fibrillation there sems to arise in the auricle a continuous shower of stimuli, which falling on the node excite it to send stimuli to the ventricle as rapidly as the bundle(and so the ventricle) is capable of taking them up. At first the ventricular contraction is apt to be very rapid, and the patient may soon die of heart failure. But if the ventricle can be made to beat more slowly, the patient may lead a useful and even vigorous life for some years. It is therefore very important to diminish the rate, and this

is done by digitalis in a remarkable manner. The gravest sign is an increase in rate; say from 100 to 150. The digitalis should then be pushed until there is a fall to 80. My experience tallies with Lewis's view; for in the permanently irregular pulse relief only comes from the continuous use of digitalis or strophanthus. Hering recognizes this fact. Mackenzie believes, however, that a good deal can be done for the patient. He finds, for example, that the irregularity is most often associated with rheumatic hearts, usually those of mitral stenosis, and in the fibroid heart of senility. In fact, as the deposit of fibroid tissue is common in both these varieties of cardiac disease, it seems possible that the fibroid deposit is the cause. In one case of mine there was a fibroid tumor of the uterus, but its removal did not improve the cardiac difficulty.

Fig. 55—Auricular fibrillation with partial heart block in a male with Adams-Stokes disease. Pulse -30. Time markings in seconds and tenths of seconds.

According to Lewis, auricular fibrillation constitutes at least 50 per cent. of all irregularities, the disturbance of cardiac rhythm having its origin in the auricle, and being due to temporary or permanent incoördination of the musculature of this chamber. In a study of 106 cases, he has reached the following conclusions, which differ somewhat from those of Mackenzie. The rate of the pulse may be reduced as low as 30, or increased to as high as 200, but this in itself has little significance, because many beats of the heart may not reach the radial artery. But the fast rates, viz., between 110 and 150, are the most common, and with these the irregularity is greatest. According to Lewis, the diagnosis rests on these points: 1. The absence of the normal auricular contraction as seen by the absence of the wave in the tracing; 2. The presence of a ventricular beat having its origin in an impulse received from the auricle; 3. Special oscillations in the curve, which have been shown to be due to the continuous contraction of the various parts of the auricle quite without system or coördination; 4. Constancy of this picture from patient to patient in respect of the three first points.

Fibrillation is responsible for most of the disturbances of the ventricular system. Indeed, in the vast majority of instances a sphygmogram showing that no two successive heart beats are of the

same length means the diagnosis of auricular fibrillation. To Cushny, Mackenzie, Wenckebach, Rothberger, Winterberg and Lewis the credit of the discovery is due. Janowski has known such a case to last five and a half years, Mackenzie ten years, I eleven and a half years.

Of the existence of auricular fibrillation there seems to be no doubt, but we are still lacking in an agreement as to the criteria necessary for its detection.

In affections of conductivity the normal stimulus, which starts in the sinus venosus, passes from the primitive tissue of the auricle over the bridge of His to the ventricle, may be delayed in its course, may not cross at all, or may be arrested beyond the bridge. Any

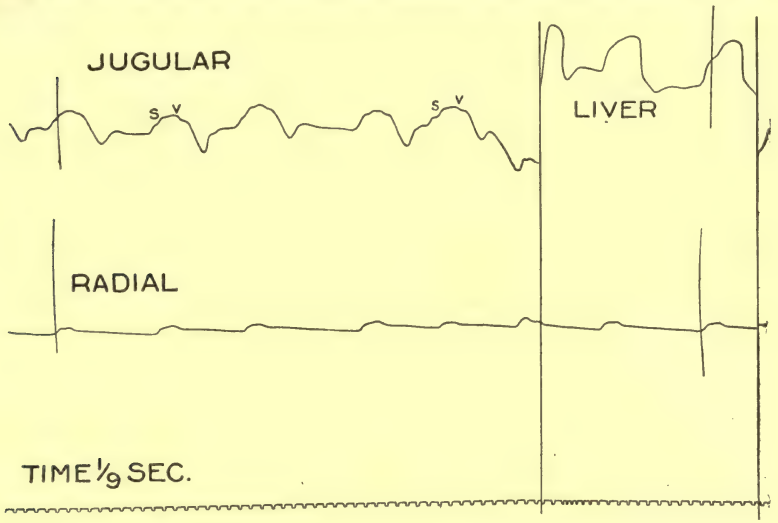

Fig. 56—Jugular tracing of what is now called auricular fibrillation, formerly called by Mackenzie nodal rhythm, in a patient with a double mitral lesion, tricuspid regurgitation, and auricular paralysis, in so far as that the apex beats are irregular, while the auricular waves are absent. *S*, is Mackenzie's auricular wave *A*, and *V* is Mackenzie's carotid wave *C*.
This tracing was taken for the author by Dr. T. B. Barringer, Jr.

one of these several conditions will produce heart block, a term invented by Gaskell in 1882 to indicate arrest or blocking of the impulse normally conducted from auricle to ventricle. In Fig. 6 is an illustration of heart block as shown in a polygram.

The pulsus infrequens, improperly called the slow pulse, is pretty certain to indicate a loss of conductivity. When, as happens in such cases, a pulse is found where only about 26 to 40 beats can be recognized at the wrist, an inspection of the jugular will usually show that the auricular contractions are really twice or even more

times as frequent. This inspection should always be made, when, if the head is turned to the left, the beating of the vein can be seen in a good light, and the diagnosis made without the aid of graphic methods.

In health the *a-c* intervals show in the graphic tracing the time occupied by the passage of the blood from the auricle to the carotid, which is usually one-fifth of a second, though it may be two-fifths, and persist at that rate for years. In complete heart block the *a-c* interval varies so much that the auricle may be said to beat quite independently of the ventricle, as in Fig. 57.

In Fig. 57 there is complete heart block. The *a-c* interval is seen to vary so that no two have the same length. Also for the 64 auricular pulsations there are only 38 ventricular, or about the ratio of 7 to 4. Dr. Manges (*N. Y. Med. Record,* 1911, LXXIX. 651) has recently given the history of a patient with incomplete and subsequently complete heart block, where the auricular rate, as shown by graphic tracings, was 280 to the minute, the ventricular ranging from 40 to 70, which rate, however, was raised to 120 on two occasions by the use of atropin.

Among the more frequent causes of loss of conductivity are syphilis, fibrosis, and neoplasms. Owing to the cause of auricular fibrillation being the same as that of heart block, though usually in the substance of the ventricle, the one may pass over into the other. But the disease may be due, and in several of my cases has been due, to hemorrhage at the base of the brain, when pressure is brought to bear on the nucleus of the pneumogastric. Stimulation of the peripheral branches of the pneumogastric may also produce heart block; also aconitin, adrenalin, muscarin, physostigmin, and aphyxia. In a case of acute heart block, lately reported to me by Dr. Waitzfelder of New York City, the condition was evidently due to overdosing by digitalis.

According to Mackenzie, the stimulus may go through and the pulse be as high as 70, and then fail to go through, leaving the pulse at 30. Such cases should encourage us to use appropriate remedies to restore the heart's action. Certainly if there is the slightest suspicion of advanced syphilis, antisyphilitic remedies should be pushed to the limit.

It is important to know that a diagnosis in heart block can be made without the use of graphic methods. At present we recognize four forms of this disturbed conductivity:

1. Acute heart block, due usually to the misuse of drugs.
2. Partial heart block, where the stimulus is occasionally carried

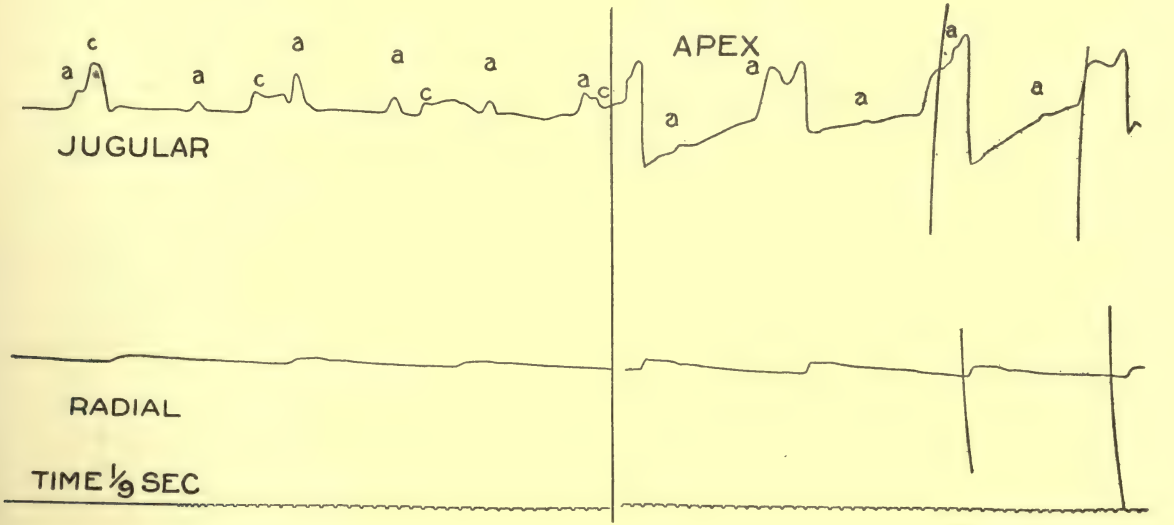

FIG. 57—Adams-Stokes Syndrome with complete Auriculo-Ventricular Dissociation (Heart-Block). This patient suffered from attacks of vertigo and syncope due to entire loss of auricular-ventricular conductivity. In the jugular tracing the waves marked *a* represent auricular contractions, and those marked *c* the carotid pulse. There are 64 auricular contractions per minute, and 38 ventricular. The auricular rhythm, as shown in the jugular and apex tracings, bears no relation to the ventricular rhythm, so that the case is one of complete heart-block.

This tracing was taken for the author by Dr. T. B. Barringer, Jr.

through, as shown by an alternation between the normal rate and the slow rate.

3. Complete heart block, where the auricle and ventricle contract independently.

4. The Adams-Stokes syndrome, where loss of conductivity is associated with syncopal attacks. The syncopal attacks are often attributed to cerebral anemia, induced by the slow action or temporary suspension of the ventricle's action.

As for Gaskell's fifth mentioned attribute of cardiac muscle, tonicity, it may be said that the loss of it is exemplified in the dilated heart.

In the matter of treatment, in (1) disturbed rhythmicity, the first of the types of irregularity that were discussed, it is quite evident that as a rule it does not warrant us in sounding notes of alarm. In other words, it is physiological, as for example in the so-called youthful type of irregularity.

In (2) extra-systole there may be a neurotic base, or it may result from the pressure of gas caused by gastro-intestinal fermentation. Or it may be a reflex from the gastro-intestinal tract, as in indicanuria. In the one case, sedatives, such as camphor or the bromides, are useful; in the latter, remedies that regulate stomach or intestinal digestion, such as pepsin, pancreatin, bismuth, sodium bicarbonate, etc. If the extra systoles are from over use of coffee, tea, or tobacco, the therapeutic indications are evident.

In (3) auricular fibrillation there is no remedy so satisfactory as digitalis or its congeners, of which strophanthus is next best. Only the most reliable alkaloids of these drugs should be used, because if given in suitable doses they are effective without causing any of the unpleasant effects usually associated with these drugs in other forms. The intelligent use of baths and resistant exercises is also very valuable.

In (4) heart block, if acute and caused by digitalis, the use of the drug should of course be suspended at once. If syphilis exists, antisyphilitic remedies should be used according to our established rules, and in sufficient quantities. Atropin, 1-60 grain, is available as a test to determine whether the heart block is due to a lesion of the pneumogastric; it will usually increase the rapidity of ventricular contractions, when these are below the normal, but it has no curative effects.

Chapter VII.

HIGH FREQUENCY CURRENTS IN ARTERIOSCLEROSIS.

The high frequency current now most generally used in and about this city is generated mainly by the combined apparatus of three Americans,—Benjamin Franklin, Morton, and Tesla; the latter, however, a foreigner by birth. In 1730 Franklin was the first to construct the influence machine, now known as the static, by which a current is derived from revolving glass plates. Although an electric current may also be obtained from chemical batteries (the discovery of Volta in 1896), from induction coils (discovered by Faraday in 1831), or from an alternating current dynamo, the static is generally regarded with the most favor.

In 1881, Morton was the first to generate high frequency currents, which he derived from the static machine and two Leyden jar condensers, to be improved in 1887 by his "step-up" transformer, where solenoids replaced the regular external armature of the Leyden jars, and further improved by the spark-gap circuits of Lodge, now everywhere in use. D'Arsonval's solenoid followed in 1891. Morton's method consisted in using an induction coil in place of the static machine. The terminals of the two Leyden jar condensers were attached to the ends of a short coil made with a few turns of heavy copper wire. One electrode was connected with one end of the short coil, and the other was brought into sliding contact with any desired turn of coil. The short coil was used to increase the induction. D'Arsonval made the discovery that a current of from 5,000 to 10,000 alternations each minute could be made to traverse the human body without doing harm while increasing oxidation, diminishing excitability, and lowering arterial tension. Apostoli and others confirmed the discoveries of d'Arsonval subsequently. (*Gazette de gynécologie,* 1895, X. 246.)

In 1891, Tesla, by means of "alternators" and "transformers," was able to further increase the potential, so that currents of higher frequency could be made not only to pass through the human body, lighting up a 100 volt 10 c.p., incandescent lamp in the circuit, but generating a current of 10,000 alternations each second. He used the induction coil, and in fact the d'Arsonval method, except that he added a secondary coil of fine wire, and a single Leyden jar condenser. He held that the greater the frequency of oscillation

the greater the electrical energy that can be passed through the body, and the less the sensibility to it.

In 1893-4, Oudin devised his "resonator," consisting of a solenoid or spool with an insulated copper wire fifty to seventy-five yards long wound around it. This instrument, when "tuned" so as to harmonize with the oscillations of the current, augmented their frequency.

The principle of the high frequency current is that if a very high potential is discharged into a conductor which has a certain self induction and a slight resistance, extremely rapid isochronous oscillations result. There may be hundreds of millions of them in each second. The ordinary alternations of a Ruhmkorff coil are about 200 a second, with an electromotive force of 10,000 to 200,000 volts, while in the high frequency currents the voltage may be from 100,000 to 1,000,000. When the current, from whatever source it has been derived, is acted on by a Ruhmkorff coil, it develops high tension. If further acted on by a spark-gap, and transformed by the condenser,—*i.e.*, the Leyden jar,—it becomes a high frequency current. The condenser augments the intensity of the current.

In 1901, my attention was first called to the beneficial effects of high frequency currents in arteriosclerosis. I then had under my charge a physician who was recovering from a mild cerebral hemorrhage, which had occurred about a month before. He had some paresis of the lower extremities and complete paralysis of the left masseter muscle, with severe muscular pains. He gradually regained the use of his muscles, and when seen by me ten years later had experienced no return of hemorrhage. But he had from the first an infrequent pulse, from 30 to 40 continuously. He had been treated by acetylparamidophenol salicylate (salophen), acetylsalicylic acid, and morphine for his muscular pains, strychnine, nitroglycerin, suprarenal extract, digitalin, cactus, and arsenic for his cardiac weakness, but in addition had a pretty extensive course of electricity under Dr. E. B. Perry, of New York, and there was no doubt in my mind but that the electrical treatment was of both temporary and permanent benefit. Dr. Perry used static electricity, with long sparks and the fine needle spray. At first the séances were daily for ten minutes; later, as improvement was becoming marked, on alternate days.

In another recent case of mild arteriosclerosis with aortic disease and cardiac hypertrophy in a married lady of sixty-five, the Nauheim treatment was given, then a course of iodine, and subsequently

the high frequency current. Dr. M. W. Johns, of Utica, New York, carried out the latter part of the treatment. Previous to her coming under my care, her maximum pressure had been 200 mm. Hg. When the electrical treatment was begun, on February 17, 1909, it had fallen to 185 mm. On March 27th it had fallen to 165 mm., on April 10th to 150 mm., and on April 17th to 145 mm. These treatments were given three times a week, each séance lasting from fifteen to twenty minutes. Her husband, a well-known physician, in reporting on her condition to me subsequently, said that as a result she was "in every way better; slept well, was less nervous, and felt much happier."

Unless one has a static machine a high frequency coil is best adapted for the high frequency treatment of arteriosclerosis. In using the static machine, there should be a revolution of the plates of at least 350 to 400 to the minute. The outsides of the Leyden jars should be connected to a d'Arsonval solenoid, or an Oudin resonator. In using the d'Arsonval solenoid, the patient is to be connected to both poles, by means of a fibre seat, one wire going into it and the other wire connected to a metal electrode placed in the patient's hands. In using the Oudin resonator, only one pole is used, a wire running from the binding post in the resonator to the fibre seat in the chair, the Oudin in this way giving the same high frequency as the d'Arsonval, but a much higher voltage. This treatment should last fifteen to twenty minutes, after which the glass vacuum electrode, attached either to a solenoid or resonator, is applied to the patient, using a spark-gap that will not be painful to him. This treatment is given from two to three times a week, for about six weeks, or until the blood measure falls to the normal or nearly to it. This was the form of treatment used by Dr. Johns, of Utica.

Dr. John P. McParlan, of New York, who has assisted me in the management of some of my cases, uses a twenty-plate machine with an Oudin resonator. He uses a current of from 200 to 250 milliampères for twenty to twenty-five minutes. His autocondensation isolation platform, fitted with a metal back, is covered with heavy plate glass, on which are laid three thicknesses of felt. On this the patient reclines. One pole of the resonator is attached to the metal back; the patient holds the other in both hands. A good method is as follows: The patient is first subjected to the high power incandescent lamp for a few minutes (Fig. 59). The blood is brought to the surface, and a sedative effect is produced. Then

the static breeze may be given. Pull the sliding poles apart, so that there will be no spark, connect the negative side of the machine to the isolation platform by the long brass shepherd's crook, grounding the positive pole. Place the metal standard near, with the crown (Fig. 60) over the head—the negative electricity streams over the patient's face, so that he feels the breeze. There is also an odor of ozone, which gradually fills the room; the oxygen of the air has been changed into ozone, or, in other words, electrified. This modality is very soothing and helpful in nervous conditions.

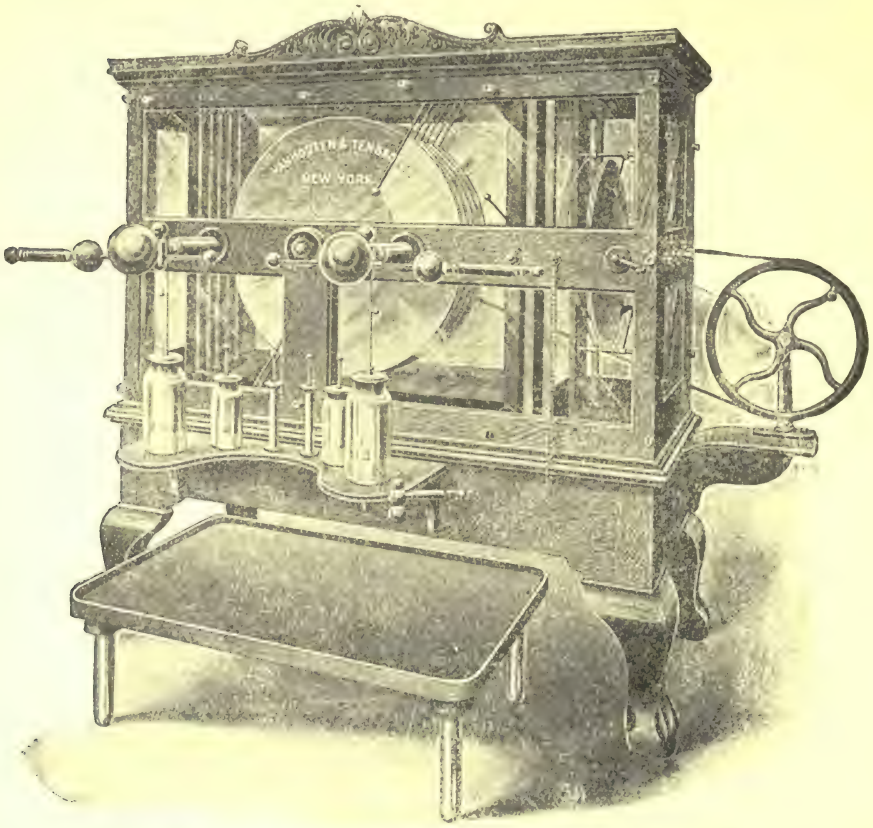

Fig. 58—The Morton-Wimshurst-Holtz influence machine.

Treatment, five to ten minutes. The patient is then subjected to the high frequency current. The plant in this case consists of a resonator, combined with a d'Arsonval solenoid, an adjustable spark gap and a pair of condensers of the Leyden jar type. I use the pattern known as the inductoresonator (Fig. 61). This apparatus is

operated by a static machine. The patient reclines on a "condenser couch," which is isolated by glass feet. The cushion has on its under surface a fine metal plate, which runs its entire length and is connected with one pole of the Oudin resonator. The other pole of the resonator is connected with a vacuum electrode, and the fluorescent spark discharge is applied through the clothing to the body

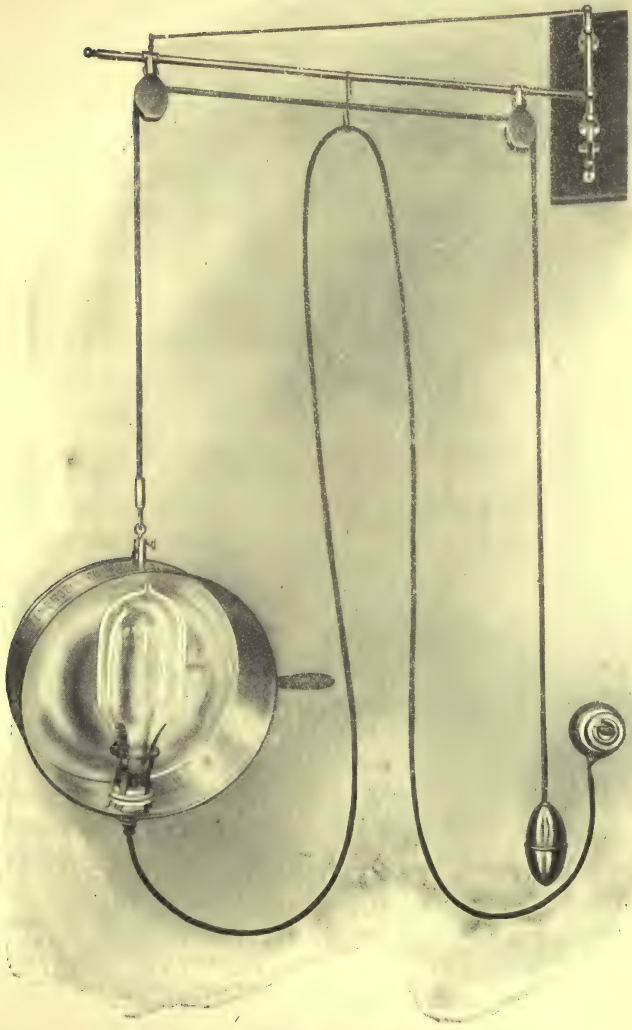

Fig. 59—High power incandescent lamp.

by the operator slowly moving the electrode over the surface. The séance lasts ten to fifteen minutes.

Static electricity is now very generally used in this country, for owing to the dryness of our climate it is easy to excite the apparatus. Most use the Holtz machine, and often inside of it is a small Wimshurst to start the larger Holtz. After the Holtz has generated a sufficient amount of current, the Wimshurst is no longer needed. The Holtz static machine will give a current sufficient for eight modalities, *i.e.,* methods of treatment (Fig. 58).

Static currents are of relatively high voltage (pressure), but the ampèrage (quantity) is small, so that you cannot burn your patient with it, if the electrode is kept moving. With this caution, therefore, you never can use too strong a current. The relative voltage is measured by the length of the spark in the spark gap, between the sliding poles. It is at least 35,000 volts for the first inch of spark, and less for every other inch. The voltage capacity depends on the diameter of the revolving disks, and the speed of the machine.

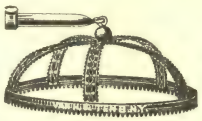

Fig. 60—Metal crown.

The Wimshurst-Holtz machines should be run at high speed, giving a current of about 150 to 500 milliampères. It is held generally that the static machine is more sedative than the coil, and in other respects more effectual.

The powerful waves of the static form of electricity cause short waves of vibration in the capillaries, promote metabolism in an extraordinary way, and do no harm. In my experience, a diminution in arterial pressure is the regular sequel to the use of the high frequency current, but the séances should not be protracted beyond the time at which the maximum pressure falls to the normal. A reduction beyond this point might, of course, be harmful.

But the high frequency currents alone are not uniformly permanently effectual. Moutier very properly states that while high frequency currents reduce arterial pressure, they are not uniformly effective, and may be inhibited by errors in diet, constipation, or various reasons. Though, as a rule, he was able to secure a reduction in the arterial pressure after a single sitting, occasionally it required as many as sixteen. The total effect, however, was pro-

duced in two, three or five minutes. But he also said that the reduction once obtained can be retained, at least for as much as three years, without relapse. My experience thus far has been that while the pressure may not always be reduced at each sitting, there is, notwithstanding, a progressive fall, in all instances, as the weeks go by.

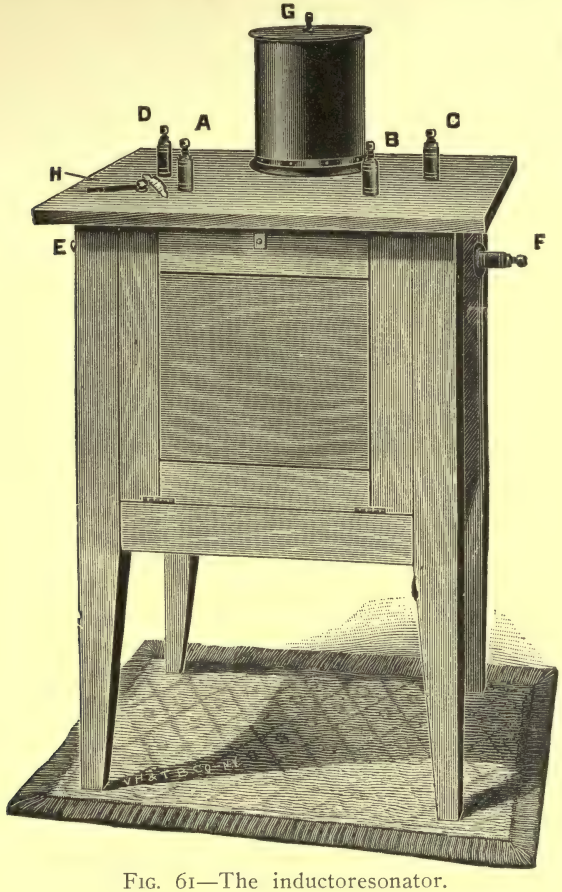

Fig. 61—The inductoresonator.

In this connection, it has sometimes been advised to use the faradic current as an adjuvant. Intestinal atony is, curiously enough, often a complication of arteriosclerosis. Then the faradic current applied over the abdominal walls may be very helpful to relieve the constipation. In addition, I use as the case indicates mercurials, iodides, and iron. In syphilitic arteriosclerosis, mercurials or the mixed treatment are surely indicated. In gouty ar-

teriosclerosis, iodides, iron, and arsenic. I am in the habit of giving them serially. A course of the iodides is to be followed by a course of iron. In very old people, I find small doses of arsenic are even better than iron.

Chapter VIII.

THE USES OF CARBON DIOXIDE.

The chief advantages of the Nauheim bath are that it contains carbonic acid, which slows the pulse rate, and at the same time regulates the circulation. This it does by dilating the capillaries and smaller vessels, and by directly and indirectly stimulating the nervous centers, while the peripheral nerves are soothed. The result is that local congestions are dissipated various inflammatory deposits absorbed, and there is improved metabolism, as shown by an increased secretion of urine, all of which acting together tone up the unhealthy tissues.

It is clear, therefore, that while carbonic acid is especially applicable for chronic heart diseases, associated with heart weakness or failure, and also for neuroses, it offers advantages in subacute or chronic joint diseases where there has been a deposit, lithemic or tubercular. Indeed it was for gout and rheumatism, more particularly, that the Nauheim bath was first used.

Now, however, it is applied for such female ailments as metritis, salpingitis, chronic peritonitis, and after larger gynecological operations, especially where there are extensive inflammatory deposits, as in the pelvic or abdominal cavities. At Franzensbad, in Bohemia, where all the springs are highly carbonated, and where I have spent several weeks for many summers, a large proportion of the women patients resort there for these complaints, and from the fact that many are sent from a distance by their physicians, and have told me of the benefits received, I am disposed to think that the carbonated waters have had much to do with the results.

One of the later uses of the carbonated waters is for neurotic cases. This has been a newer feature of the Neuheim cure. And my experience at the Manhattan State Hospital for the Insane on Ward's Island, of carbonated baths in the treatment of mania, under the late Superintendent Dr. Dent, was very convincing as to their efficacy. Patients who previously had been quieted by drugs only, were simply kept in a carbonated bath, hour after hour and indeed sometimes day after day, under the care of a skilful attendant, and relief of the symptoms was pretty certain to follow, without any other form of treatment.

Of course, carbonic acid, or carbon dioxide, as the chemists

prefer to call it, has been known in medicine for centuries, but the profession has given it comparatively little recognition. In fact, it is not now included in the last U. S. Pharmacopeia, nor is it in any materia medica I have consulted, though a good deal of space is given to it in Stillé's and Maisch's National Dispensatory.

As a component of the numberless cooling draughts, we enjoy it, but in medicine it has been almost entirely supplanted by other remedies. And yet it was once used extensively as a local application for mucous membranes; also as an anti-emetic. In the last half of the nineteenth century, Percival found that it was a disinfectant, and analgesic, and improved nutrition. At one time it was employed with considerable success for ulcerating cancers, where it was found not only to relieve pain and destroy odors, but improve the patient's condition generally. It was also at one time in the last century a popular remedy for phthisis, especially in France. As late as 1883 Weill gave it by inhalation for phthisis. The cough was relieved, but only by its narcotic qualities, so that this method was soon abandoned. A few years later, in 1886, Bergean injected it into the rectum, intending it to reach the lungs, through the venóus system, without affecting the arterial. To the carbonic acid gas he added some sulphuretted hydrogen. His aim was to kill the tubercle bacilli in the lungs. The method was of course ineffectual. The gases never reached the lungs. Better results, however, were gained in spasmodic asthma and whooping cough. The gas was also, in these affections, injected into the bowel, and it reached, as was proved, the ileo-cecal valve. By inflation of the bowel in the afternoon, night attacks of asthma were prevented. Professor Rose has reported good results and indeed permanent cures in such cases. The same method has been attended with good results, it is said, in dysentery and membranous enteritis. As a soothing application for various mucous surfaces carbon dioxide has also been used. It is still a remedy at Ems for impotence and torpor of the genito-urinary organs, probably stimulating the nerve centers while it acts as a local sedative. In typhoid fever one or more physicians of this city have preferred the carbonated bath to the ordinary cool bath. With a temperature of 90 deg. F. it has been thought to be both tonic and sedative and better borne by the patients than the Brand bath. In this way it has been used at Roosevelt Hospital.

One of the oldest methods of using carbonic acid is by the dry plan, and there is a spring at Franzensbad in Bohemia known as the Polterbrunnen, where the gas is collected as it emerges from

the ground, and is conveyed into a building where there are chambers just below the surface of the ground. Patients take this dry gas sitting in chairs and without removing their clothes. The gas being heavier than the air rises only a few feet, so that there is no danger of inhaling it. Soon after the gas has been turned on, there is a sensation of tingling in the feet and legs, up to the level of the gas, and subsequently an agreeable sensation of warmth, all due to dilatation of the capillaries. Persons who object to the Nauheim bath can take the gas in this dry form. The effect in each case is similar in kind, so far as the gas is concerned, but the warm water, presence of common salt and chlorid of calcium intensify the action of the gas.

Professor Rose, of New York City, has devised a portable tub, by which this dry method can be carried out successfully at one's home.

A novel application of carbonic acid by the douche has been proposed by Dr. Bloch of Franzensbad. Recognizing the fact, discovered by Dr. Abrams of San Francisco, that friction of the precordial region will produce contraction of the heart, he proposes to douche the surface with carbonated water, by which he aims to produce the joint effect of friction and of carbonic acid. Theoretically this method should be an advance, if given cautiously and in suitable cases.

The action of the Nauheim or carbonated bath is due mainly to three things: Carbonic acid, chlorid of sodium, and chlorid of calcium. Carbonic acid dilates the capillaries directly, and also in a reflex manner, so that the peripheral resistance in the blood vessels is diminished. The first effect is a slight increase in the tension of the vessels, followed by a long continued lowering of it.

Sodium chlorid apparently increases oxydization and is said to cause a 25 per cent. more evolution of gas. Calcium chlorid acts chiefly as a skin irritant, intensifying the slightly irritant action of the sodium chlorid, and probably favors tissue metabolism somewhat.

In our estimation of carbonic acid we should remember that it is a normal constituent of the body. Entering it as carbon, it is converted by an excess of oxygen into carbonic acid; or urea, which in decomposing gives off carbonic acid. It also exists normally in the blood, being one of its principal gases, the others being oxygen, nitrogen and perhaps argon. In arterial blood carbonic acid is about twice as abundant as oxygen, being present in about 30-40 per cent. by volume, while oxygen is only present in about

20 per cent. In venous blood, on the other hand, there is a larger amount of carbonic acid, about 48 per cent. by volume, which in cases of asphyxiation may reach nearly as high as 70 per cent. In arterial blood about all the oxygen present is found in the red corpuscles, while less than one per cent. is in the serum. In venous blood, on the other hand, the serum contains the largest amount of carbonic acid, while perhaps one-third or so is in the corpuscles. In them it exists in loose chemical union with the alkaline phosphates and hemaglobin.

Carbonic acid gas is colorless, non-inflammable, and absorbs at the ordinary temperature about an equal amount of water. But it is heavier than air, and may be converted by pressure into a liquid and even into a solid white mass.

So far as the danger of inhaling carbonic acid is concerned, there need be little fear. Even 3 per cent. produces no permanently deleterious effect; though 5 per cent. causes dyspnea, and 10 per cent. narcotism; 1 per cent. however is almost insufferably disagreeable, and yet it is recognized so easily by its peculiar odor that one is forearmed When suspended in the water it is at most barely 3 per cent, and hardly appreciable to one's olfactories With the thousands of carbonated baths given annually at the various resorts, I have never heard of any cases of narcotism, even among the bath attendants, who are of all persons most exposed. At most, on a warm day and in a small room with windows shut there may be a sense of "close air," which disappears as soon as a window is slightly opened.

I am not disposed to vaunt the use of carbonated waters except in a somewhat narrow field, but I hold that as an ingredient of the Nauheim bath it has won for itself a permanent place among our newer therapeutic resources.

The methods of producing gas for the artificial bath are various. There are both machines for generating it and for mixing it. Abroad the apparatuses of Moosdorf and Hochhausler of Berlin, and Fischer and Kiefer of Karlsruhe are much used in localities where there is no natural carbonic acid gas in the waters.

For the purpose of determining the carbonated water in a state of minute division in the bath tub, the Kny-Scheerer Co. have devised special mixing cylinders, to be used in connection with the ordinary liquified carbonic acid drum, or cylinder (Fig. 62). The two mixers are attached together but the larger one is also connected with the liquified gas in the carbonic acid cylinder or drum, which in turn is connected with the water supply pipe. According to the

manufacturers, "The principle upon which this apparatus is constructed lies in the possibility of mixing gas and water under the same amount of pressure. The pressure of the hydrant water is the unit according to which the gas pressure is regulated. To accomplish this purpose a pressure regulator is placed upon the water supply pipe, also a pressure registering gauge. For connection with the liquified carbonic acid cylinder, a gas pressure regulator and pressure registering gauge are also supplied. This forms the equipment ready to be set up at the head or foot end of a bath tub, and the apparatus is ready to be connected by any plumber with the water supply pipe."

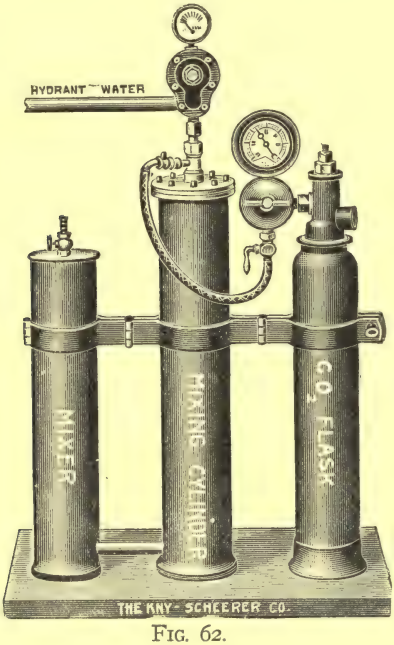

FIG. 62.

While, however, special machinery is well adapted, for economy's sake, in bathing resorts where a large number of baths are given daily, the generation of gas by the acid sulphate of sodium tablets in conjunction with sodium bicarbonate, as manufactured by H. A. Cassebeer[1] and others of this city has proved satisfactory in my hands, and I have generated gas in this way for twelve years

[1] Now made by the Shepard Pharmacal Co., 275 Water St., New York City. Cost of the ingredients for a single full bath, inclusive of the common salt, is 90 cents.

and more. Quite recently, however, a new method has been devised by a New York firm[2] (Fig. 63).

The gas is developed by adding to a sodium bicarbonate powder a solution, said to consist mainly of an admixture of organic acids, (formic, lactic, etc.) with a small percentage of hydrochloric acid. The method of preparing the bath is as follows:

An ordinary bath tub is filled with about fifty gallons of plain water at a temperature of 90 deg. The sodium powder contained in a bag is then sprinkled over the bottom of the tub, but mainly on the spot upon where the patient is to sit. As soon as he has taken his seat in the bath, the developing solution, to the amount of about oz. xx, as contained in a bottle, is poured about him. A lively evolution of gas ensues. The very trivial amount of free gas that will necessarily float on the surface of the water, is waved away by the hand at occasional intervals, so that it cannot pos-

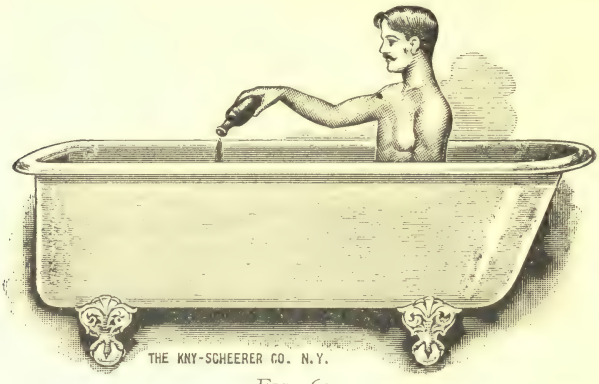

Fig. 63.

sibly be inhaled. The advantages claimed for this new method are —that the solution is stable; that being mainly composed of organic acids it cannot in any way be injurious to the patient or to the plumbing fixtures, and more gas is generated than by any other method. It has an agreeable odor. The cost of the ingredients for a single full bath is 75 cents; for a set of 12 baths, $8.00; so that the total cost is only $16.00 for an ordinary course of 24 baths. It promises well, though at present, my experience with it has been limited.

In using the acid sulphate of sodium tablets prepared by Cassebeer and others, the tablets are placed upon a rubber mat[3] laid on

[2] The Kny-Scheerer Co., of New York.

[3] Furnished by the Shepard Pharmacal Co., 275 Water St., New York City.

the bottom of the tub, to prevent corroding of the tub, in case it is made of metal or is metal-lined (Fig. 64). The rubber mat also provides a firm support for the feet, which is agreeable, and obviates the danger of having the patient's feet slip when getting in or out of the bath-tub.

The ingredients, as supplied by Cassebeer, are put up in small wooden boxes about 8x6x4 inches, containing in dry compact form eight discs wrapped in heavy lead foil and four packages of powder, sufficient for one bath of the fullest strength required.

Both elements are almost chemically pure. All the water of crystallization has been eliminated from the discs, which contain about forty-two per cent. of free acid. They are hard and do not deliquesce at any temperature, which is important. They keep an indefinite time, but being hard, dissolve slowly. By this method

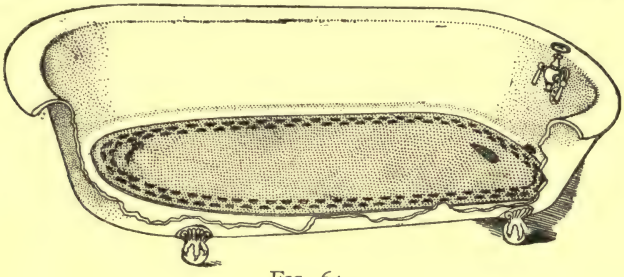

Fig. 64.

the evolution of carbonic acid gas is continuous until the tablets are dissolved, and as much of the gas is given off as there is in an ordinary carbonated bath at Nauheim.

For bathing salt it is often preferable to begin with an ordinary American sea salt, as by so doing one gets the benefit of the iodides and bromides it contains, but if this is not important, us a good quality of dairy salt, such as the Genesee. At the end of the first week carbonate the baths. Then, when a larger amount of salt is to be used, substitute ordinary bathing salt for the sea salt, increasing at the same time the amount of carbonic acid gas. When the gas exists in a fair percentage in the bath, its presence is shown by minute bubbles attaching themselves to the body of the bather. At the middle of the course fortify the ordinary bathing salt with pure calcium chloride.

The Triton Company, through Schieffelin & Co., agents, also prepare effervescent bath salts especially for the Nauheim treatment and furnish directions for their use. In using the effervescent salts my method is as follows:

PLAN FOR THE ORDINARY SIX WEEKS' COURSE

	1st Week No. 1 Bath	2nd Week No. 2 Bath	3rd Week No. 3 Bath	4th Week No. 4 Bath	5th Week No. 5 Bath	6th Week No. 6 Bath
Percentage of Sodium Chloride	½ per cent	¾ per cent	1 per cent.	1¼ per cent.	1½ per cent.	2 per cent.
Quantity of Sodium Chloride..	2 lbs.	3 lbs.	4 lbs.	5 lbs.	7 lbs.	10 lbs.
Calcium Chloride.....				6 ozs.	9 oz.	12 oz.
Percentage of Carbonic Acid Gas		¼ per cent	½ per cent.	¾ per cent.	¾ per cent.	1 per cent.
Quantity of Effervescent Nauheim Salts		2 discs to 1 pkge.	4 discs to 2 pkgs.	6 discs to 3 pkgs.	6 discs to 3 pkgs.	8 discs to 4 pkgs.
Quantity of Water.....	50 gals.	50 gals.	50 gals.	50 gals.	50 gals.	50 gals.
Temperature of Water.....	98 deg. F.	97 deg. F.	96 deg. F.	95 deg. F.	94 deg. F.	93 deg. F.
Duration of Bath.....	4 minutes	6 minutes	8 minutes	10 minutes	12 minutes	14 minutes
Intermission	3rd and 6th day	4th day	4th day	4th day	5th day	5th day

An ordinary Nauheim bath contains from two to three and one-half per cent. of salt, of which about eighty per cent is sodium chloride and ten per cent. calcium chloride.

It will be noticed that the amount of carbonic acid gas in the bath varies from one-quarter to one per cent.

The number of baths in a full course is 35, but sometimes a less number is sufficient. Twenty-four is a good average number. With robust people it may be as well to commence at once with the carbonated brine bath. Sometimes, especially with delicate persons, the number 4 bath will be as strong as is desirable.

The method of preparing the bath is as follows: Fill the bath tub, preferably of porcelain or enamelled iron, with 50 gallons of water at about 105 deg. F.; then add the ingredients as follows: first add the required quantity of bath salt, then place the discs (broken or pulverized), intermixed with the powder on a couple of saucers or on a single plate, or on the sheets of heavy lead foil furnished in each package, or directly on the rubber mat, if one is used on the bottom of the tub. An evolution of gas takes place in a minute and lasts twenty or more minutes. The patient should enter the bath when it has reached 98 deg. F. or lower, according to the directions, and remain in it the prescribed length of time. As soon as the gas is evolved it will be seen attaching itself to the trunk and limbs in the form of minute bubbles.

Recently there has been devised a false bottom (as illustrated) to be used in the ordinary bath tub in connection with the Nauheim Bath Salts. This appliance is made of heavily enamelled sheet steel, about 14x36 inches, with three supports of hard wood on the under side of sufficient height to raise it about three inches from the bottom of tub. It may be placed on the rubber mat.

In use the bath is prepared as per the directions given above and the false bottom is then placed directly over the effervescing bath salts; the carbonic acid gas as fast as generated rises through its perforations and around its sides, thus reaching the bather from every quarter without obstruction.

It will be readily seen that, while not absolutely essential, this device makes it possible to administer the Nauheim treatment more effectively and with greater ease and comfort, for both operator and patient.

The advantages of the artificial bath may be briefly summarized as follows:

It contains the chief natural constituents of the Nauheim waters, and is applicable in most chronic heart diseases. The carbonic acid

gas is held in better solution by the artificial bath than by the natural waters of Nauheim. The artificial bath is cleaner than the natural, which contains substances of no obvious utility. The artificial bath is taken at home rather than in a public resort, and just before retiring so that there is little or no danger of catching cold. An artificial bath costs less in this country than in Germany, and the total cost of the treatment brings it within the reach of a moderae purse. There is no danger from free carbonic acid gas, as it is held in closer combination than in the Nauheim waters. The discomforts of a foreign trip are avoided.

On the other hand there are certain advantages in the Nauheim natural baths. Whatever of virtue there is in the natural water is retained. The patient has the choice of numerous physicians of repute in the treatment of chronic heart disease. The patient being away from home, is for the time entirely free from many of the annoyances of business, or the cares of private life, in the comparative seclusion of a small watering place,—matters of value in the treatment. He may find the Nauheim course an agreeable feature of a summer jaunt abroad.

Whether it is better to take a course at home or abroad is for each individual to decide after comparing the advantages of one with the other, as seen from his special point of view.

So far as the treatment is concerned, it is certainly quite as well given in this country as in Europe, and with more comfort to the patient.

Chapter IX.

MOBILITY AND MALPOSITIONS OF THE HEART.

It has long been known that the heart does not always occupy a fixed position in the thorax. In fact, it moves up and down, from side to side, and under certain circumstances is rotated a little on its axis.

The degree of its lateral mobility, however, appears to have been first investigated by Cherchersky, of St. Petersburg.[1] In a total of thirty-nine instances he found that in nineteen there was a good deal of mobility; in five there was a little, while in fifteen the heart's position was fixed. He also noticed that the total range of excursion,—*i.e.*, from the extreme right to the extreme left,—was from one and five-tenths to three inches. It was his opinion that the elasticity of the great vessels at the base of the heart was the cause of the cardioptosis, which, expressed in English, means the sagging downward of the heart.

Rumpf, of Bonn,[2] following Cherchersky, claimed that if an individual assumed the left lateral posture a displacement of the apex to the left of eight-tenths of an inch, or if the right lateral posture a displacement to the right of two-tenths of an inch, could no be regarded as abnormal. In other words, a total excursion range of one inch was within the limits of normal mobility. In one instance, however, he found the total range just a little short of five and a half inches.

From an examination of a thousand persons, Pick[3] found that slight mobility was common. He believed that mobility was usually congenital, but that it might be acquired as a result of the reduction cure for obesity, or if emaciation set in. When the mobility was within the normal range, there were, in his opinion, no subjective signs, but where this limit was exceeded there might be palpitation, precordial oppression, and vertigo. In seven of his cases, where the mobility of the left varied from one and five-tenths to three inches, there was discomfort when lying on the left side. To make a brief statement of his conclusions, he found that cardiac mobility was often unaccompanied by disagreeable sensations, but that at times there were palpitation and vertigo, while after exer-

[1] Cherchersky: *Gaz. Med. de Paris*, 1887, IV. 629.
[2] Rumpf: Verhandlungen des Siebenten Congresses für Inneren Med. 1888, VII. 221.
[3] Pick: *Wien. klin. Woch.*, 1889, II. 747.

tion, or when lying on one side, particularly the left, precordial oppression was present.

Leusser,[4] in 1902, when reporting three of these cases, called attention to the additional signs of dyspnœa, pain, and even "stitches" in the præcordial region.

Previous to this, Gerhardt[5] had noted an occasional displacement of from one and three-quarters to two and three-quarters inches.

In rectifying malpositions of the heart in lateral curvature of the spine, where the organ is usually displaced to the left, the writer in one instance brought in the apex two and five-eighths inches by means of corrective exercises.[6] It must also be remembered in this connection that in forced inspiration the heart can be forced downward, while after prolonged expiration the diaphragm may move upward, carrying the heart with it. This up and down movement is well shown by the Röntgen ray, which also demonstrates that the heart does not always rest on the diaphragm. Indeed, in healthy persons it is so firmly fixed at its base that it does not ordinarily ascend and descend with the diaphragm.

An acute Italian observer named Rummo appears to have been the first to describe (in 1900[7]) a peculiar form of cardioptosis, now generally known as Rummo's disease. According to Ferrannini, of Rummo's medical clinic at the University of Palermo, his chief had described the condition several years prior to its announcement in a German journal. Rummo defined this complex as a true cardioptosis; *i.e.*, a falling or sagging of the heart, due to relaxation of the elastic supports of the organ. It was not a manifestation of Glénard's disease, for it was independent of enteroptosis or splanchnoptosis. He therefore called it primary, or essential, cardioptosis. It was found in bony persons, with a long thorax and flabby muscles. Together with the low position of the heart, he found expansion of the aorta, especially of its arch, and a tendency to hæmoptysis and varicose veins. The margin of the lungs extended below the free border of the ribs, as in emphysema, while at times the left lobe of the liver was depressed. The symptoms, as described by Rummo, consisted of præcordial oppression, anxiety, dyspnœa, palpitation, pseudoangina, slow or rapid pulse, neurasthenia, and cardiac insufficiency. This complex was thought to be hereditary, and the essential cause disease of the elastic tissues in the great veins, and, to a less extent, in the great arteries at the base of the heart. In the same year, Ferrannini[8] published four of these cases, in one

[4] Leusser: *Münchener med. Woch.*, 1902, XXVI, 1095.

of which there was epigastric pulsation. In these instances, relative dulness began either at the level of the third rib, the third interspace, or the fourth rib. There was no cardiac enlargement, but in one case there was mitral stenosis, and in another mitral stenosis with lack of physical development and deformtiy of the bones. All four cases exhibited the stigmata of degeneration.

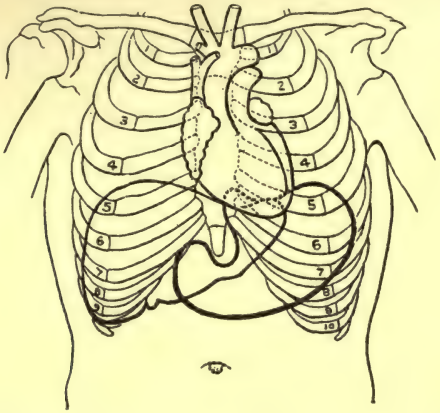

Fig. 65—Normal relations of the heart and great vessels, liver and stomach to the walls of the chest.

From an examination of 926 cases, of which 512 were men and 414 women, Einhorn found cardioptosis in 22, or 2.4 per cent.

In health, relative cardiac dulness should begin about the middle of the second interspace, whether to the right or left of the sternum. The apex should be in the fifth interspace, about half way between the left border of the sternum and the line of the chondrocostal junction (see Fig. 65).

In Fig. 66 is seen the position of the heart, liver and stomach in a patient I saw with Dr. R. C. Kemp in his service at the Manhattan State Hospital, on April 8, 1908.

A., aged 30, height 4 feet 8 inches, and weighing 80 pounds, was suffering from dementia præcox and epilepsy. She had an extreme vertical gastroptosis, shown in Fig. 66, after inflation with carbonic acid gas. The upper level of the heart corresponded with the fourth rib; the apex was in the sixth space. There was slight arrhythmia, but no valvular murmurs existed, and the heart was not enlarged. The maximum blood-pressure was 85. The upper level of the liver corresponded with the sixth rib, and the lower edge projected slightly below the free border of the ribs.

This was a case of cardioptosis associated with enteroptosis (Glénard's disease).

In Einhorn's fifteen cases of cardioptosis, an analysis gives the following results: Usually an increased mobility, ranging from

three to five inches to the left, while the mobility to the right might not be noticeable. In half of these cases enteroptosis; always some hepatoptosis. Emaciation and neurasthenia had important relations to the cardioptosis. The principal subjective symptoms were palpitation and nervous disturbances of the organ, vertigo, and an inability to sleep on the left side. When associated with enteroptosis, the heart seemed to be the first organ to regain its normal

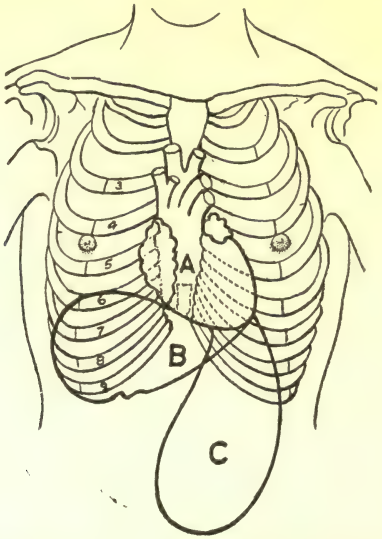

Fig. 66—Cardioptosis with the organ twisted slightly on its axis, somewhat flattened on its inferior surface, and the apex carried ot the left. The level of the heart dulness begins at the fourth rib; the apex is in the sixth space. The upper margin of the liver corresponds to the sixth rib; the free edge projects below the free border of the ribs. There is extreme vertical gastroptosis. *A,* Heart; *B,* Liver; *C,* Stomach.

position, and the return was more frequent than with the other organs.

According to Hemmeter,[9] in enteroptosis the heart is usually in an abnormal position. It is not only lower than normal, but somewhat twisted on its axis, the right auricle and ventricle resting rather flatly on the diaphragm (Fig. 66). This displacement is usually congenital, and part of an anomalous condition which has as a prominent feature an abnormal relation of the bones of the skeleton to one another. This condition of the bony framework Hemmeter expresses in terms of the infra-xyphoid angle (Fig. 67). In 638 healthy men and women examined at the age of thirty, and with a height of five feet, he found the average infra-xyphoid angle 82.1 deg.

In Fig. 67, *A* represents the tip of the xyphoid cartilage; *B* the right anterior superior spine; *C* the left anterior superior spine. The angle *BAC* is the infra-xyphoid angle, and is normally 82.1 deg., or nearly a right angle.

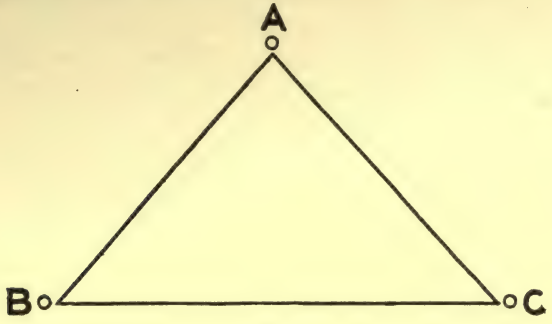

Fig. 67.

In Fig. 68, *A* represents the tip of the xyphoid cartilage; *B* the right anterior superior spine; *C* the left anterior superior spine. The angle *BAC* is the infra-xyphoid angle, and in enteroptosis averages 50.1 deg. in males and females.

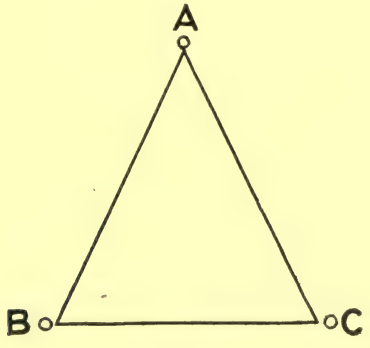

Fig. 68.

There are several other forms of cardiac displacement, one of which has two subdivisions; the intrinsic, where displacement is due to enlargement of the organ, and the extrinsic, where it is the result of such external causes as gastric or intestinal distention, fluid in the pleural or abdominal cavities, abdominal tumors, aneurisms, enlarged liver or spleen, pulmonary disease, pleuritic adhesions, a pregnant uterus, or deformity of the thorax, as in lateral curvature of the spine. In almost all lateral curvatures there is displacement;

usually to the left. I have published six of these latter cases.[10]

Of displacements downward, then, there are two varieties, (1) Rummo's disease, essential or primary cardioptosis, and (2) cardioptosis associated with splanchnoptosis (Glénard's disease). Then there are the intrinsic displacements (3) due to an enlarged heart, and extrinsic displacements (4) due to external causes. In addition, there is (5) voluntary displacement or autocardioptosis, such as is practised by individuals who can at will throw the heart from one side to the other, or move it up and down. Finally, there is (6) the congenital displacement associated with transposition of viscera, where the heart, liver, stomach and spleen have their relative positions reversed, those usually found on the right side being on the left, and vice versa.

From what has been stated, it is plain that a mobility of the heart that has a total range of not more than one inch seldom calls for treatment. When, however, it is of such extent as to cause discomfort, violent exercise and excitement should be avoided, and the sleeping posture should be on the side opposite to the one where there is the most mobility. In cardioptosis, whether of the essential form or that associated with splanchnoptosis, the diet should be carefully regulated, sugary and starchy foods being avoided, and tea and coffee minimized. Sometimes it may be better to sleep with a low pillow. An effort should also be made to restore the nervous tone of the system. According to Einhorn, it is best to increase the bodily weight; adipose tissue, he thinks, tends to restore the organ to its position, or, if it is replaced, to keep it there. An abdominal belt or supporter should be worn.

In the intrinsic form, the prognosis will be variable. The athletic heart will always be large, but the fat heart of obesity may be reduced in size. Dilatation of the heart can be relieved by rest and the intelligent use of drugs, baths and passive exercises. In the extrinsic forms of displacement, the underlying causes must be recognized and treated. And it is especially important that in the cardiac displacements due to lateral curvature of the spine there should be systematic and regular gymnastic exercises. They always give relief, and should invariably form a part of the daily routine.

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Chapter X.

CARDIOVASCULAR THROMBOSES.

Thrombosis may occur either in the heart or in the blood vessels, and from different causes. The cardiac thrombus is usually found in the auricular appendages or inter-trabecular spaces, but ante-mortem thrombi occurring in the heart are rare. It is true that before post-mortem examinations were made by experts, intra-cardiac thrombi were often supposed to be the cause of death; but in a personal experience that has certainly covered more than a thousand post-mortem examinations,[1] I very rarely attributed death to this cause. I am willing to admit that in a few instances death may have been due to a detached thrombus or portion of one, as in one case where death was ascribed by me to an arterial plug, with associated thrombosis in the Pons Varolii. But in the vast majority of instances such accidents are due to detachment of vegetations from the surfaces of diseased valves. In the cardiac insufficiency of myocardial diseases clots often form, but, as with the vulvular vegetations, they usually lodge in some part of the system where the plugging of the artery is not of sufficient moment to cause much disturbance, owing to the prompt formation of a venous collateral circulation. Most,—in fact the very great majority,—of the clots found in the heart at death have formed in the agonal stage or after death.

To the expert eye the differentiation is comparatively simple. Whatever may be the color and consistency of these late formations, the ante-mortem clot is very generally of a muddy-brown color, rather dry and friable. Often it has begun to disintegrate. Of course such a clot, certainly if large in size, has been an embarrassment to cardiac action during life. This subject is one that is only now beginning to be seriously considered by pathologists and clinicians. The case published by Dr. George Dock, to be described, will bring the matter into such notice that clinicians will perhaps be able to recognize the condition in time to come, certainly if the thrombus is in the right auricle.

So far as I know, this instance is the first on record in which even a probable diagnosis was made ante-mortem. (Smithies, *Journal of the American Medical Association*, 1909, LVI. 1347.) The

[1] I have in my possession the records of 915.

patient, a negro forty-three years of age, was found on examination to have moderate cyanosis, and a small, weak, irregular pulse. The heart was enlarged, and there were abnormal sounds at all the valves. After treatment the abnormal heart sounds disappeared, but later the previous symptoms returned. Two days before the patient's death it was observed that the heart had enlarged greatly, in its transverse diameter, and that the enlargement corresponded with a dilatation of the right auricle. A suspected thrombosis of this auricle was entered in the clinical records of the hospital. At the post-mortem examination there were found "currant jelly" thrombi of both auricles, the right being enormously distended by a blood clot. The right margin of this auricle had extended "four fingers' breadth beyond the right sternal margin." The clot extended through the tricuspid valve into the right ventricle.

How far treatment in such cases may be successful is a matter of conjecture. Certainly the condition is one calling first of all for prophylaxis, and heart tonics of sufficient power should be given to overcome the cardiac weakness, and kept up as long as needed. Digitalin, strophanthin and glonoin can usually be given continuously for weeks and months at a time without any disagreeable secondary effects, and are appropriate in such instances. This has been my experience in myocardial cases. It is also desirable to have cultures of the blood taken to determine whether the thrombosis may be due to infection. If this be present, the particular serum corresponding to the infective agent may be given. A fairly large number of cases have been reported in which some success has followed the use of appropriate sera in blood infection; but, on the other hand, the vaccine treatment or injection of dead bacteria of the same type as those found in the blood has often been inefficacious, if not actually harmful.

Arterial thrombi are not so common or so widely distributed as venous thrombi. They occur where the vessel is dilated or contracted, and on uneven surfaces, as in arterial degeneration, where the inner coating of the vessel is rough or eroded. In the veins, thrombi form in the pouches, round about an embolus, or where smaller veins open into larger ones; also as gradual prolongations of thrombi from small veins pushing forward toward the heart. Gould (*Lancet*, 1902, I. 1583), thinks they are formed in cases of sudden enlargement of veins, or when there is a "regurgitation" from the deeper to the superficial veins. Pressure, traumatisms, neoplasms, and infective processes due to pus-producing bacteria, possibly also to other micro-organisms, are etiological factors.

When thrombi occur in an artery or a vein, the vessel is apt to become obliterated. This is particularly true of the femorals, renals, portals, and the Venæ cavæ. At first the clots are soft and moist. If benign they contract and become drier, and may be absorbed sufficiently to allow the passage of the blood current. Or the clot may undergo calcific change, forming in an artery an arteriolith or in a vein a phlebolith. It is a favorable sign when a clot begins to harden, as it may then disappear wholly or in part. But the prognosis is unfavorable when the clot breaks down so as to form an abscess, though even then the incident may have only a local significance, as is the case with the abscesses and ulcers that form in the clots of varicose veins of the leg. If, however, a large clot should be driven into the pulmonary artery, sudden death would probably be the result.

The most favorable sequel is when the clot is replaced by connective tissue, which in this instance is developed from the wall of the vessel, the clot itself gradually disintegrating. Subsequently the vessel is replaced by a cord of the new tissue, and its function as a blood vessel is lost forever.

The character of the change in arteries is much like that in veins, but in the latter a collateral circulation is established, whereas in the plugging of an artery there is death of the tissue supplied. Occasionally, however, in certain organs, the function of the vessel is compensated for. In the kidney, for example, after chronic inflammation the new tissue of the thickened capsule contains arterial twigs that aid in sustaining the nutrition of the organ when its normal arterial supply is defective.

The uncommon existence of venous thrombosis with cardiac disease was brought prominently before the profession in 1900 by Dr. W. H. Welch. (*Trans. of the Amer. Phys.*, 1900, XV. 441). He found from his own experience and that of others that in cardiac diseases associated with either infective or chronic wasting diseases, such as influenza, typhoid, cancer and tuberculosis, there was sometimes a venous thrombosis, particularly of the left upper extremity. He gave a summary of 28 cases, the results of his analysis of which were as follows:

Out of 24 cases, there were 17 females and 5 males, the sex of 2 not being stated. The age varied from 9 to 53 years. In 21 out of the 28 cases there was mitral disease, and in 12 the disease was confined to the mitral. About half the cases has a previous history of articular rheumatism, but other diseases were possibly causal agents. In most instances the mitral disease was advanced, and the attack

came on during a period of cardiac insufficiency. In 22 out of 24 the thrombosis was on the left side, usually in the left upper extremity. Disease of the veins was supposed to play only a minor part in the etiology. The local symptoms were redness, pain and tenderness.

Of 24 cases 20 ended fatally; 4 recovered. The cardiac disease was the cause of death, not the venous thrombosis. From an analysis of 44 thrombi made in Dr. Welch's laboratory, and from the discovery that in 34 there were bacteria, Dr. Welch was led to feel that infection of the blood was the cause of the thrombosis.

In the discussion which followed, two cases were alluded to, one by Dr. Francis P. Kinnicutt, which had not terminated fatally (case 29), the other by Dr. Carey (case 30). Case 31 was reported by Dr. A. A. Smith, of New York. (*Med. Rec.*, 1900, LVIII. 72) Case 32 was reported by Dr. J. A. MacGregor. (*Am. Med.*, 1901, I. 353.) It occurred about six weeks prior to death, during broken compensation, and involved the left axillary, subclavian, internal jugular, and possibly the left innominate. Cases 33, 34, and 35 were subsequently reported by Dr. Frank Billings, of Chicago. (*Gen. Med. Pract. Series*, 1901, I. 117.) In two out of these three the cardiac disease was myocardial.

Case 36 was reported by Dr. J. W. Brannan. (*Med Rec.*, 1902, LXI, 291.) A woman of forty, who had suffered repeatedly from rheumatism, was admitted to hospital with evidences of valvular disease, fluid in the right pleural cavity, scanty urine containing albumin and casts, and edema of the entire body, but especially of the neck, right shoulder and arm, and the right side of the body as far down as the umbilicus. The patient survived nine days. Prior to her death various opinions were expressed as to the cause of the thrombosis. At the autopsy there were found thromboses of the right subclavian, right axillary, and right brachial. There were no thrombi in the jugular. Thrombosis ended abruptly at the right innominate. The superior vena cava was free, but the right auricle contained a pink thrombus. All the valves except the pulmonary exhibited signs of acute endocarditis, and all the heart cavities were dilated. It was suggested that perhaps the right-sided pleural effusion might have been a factor in the production of the thrombosis.

The following case (No. 37) I report. A lady past middle life, a neurasthenic and inclined to obesity, had experienced an attack of heart failure at a time when she was traveling in a mountain region. Three months prior to my seeing her in consultation she had felt a sudden sharp pain in one of her legs. At the time I saw her, she

walked with apparent difficulty at times and again with ease, suggesting the intermittent claudication of Charcot. Spasms of the muscles of the legs were said to follow walking, or even excitement, and then complete disability. The heart was found enlarged, without valvular murmurs. The heart sounds and pulse were feeble. There was thrombosis of the left upper extremity, following embolism, it was thought, the latter having caused the sudden pain. The patient was treated by heart tonics, baths, and exercises. She is now able to go about much like other people.

In the majority of these cases the thrombosis is in the left upper extremity, but it may be elsewhere. From an analysis of the cases here given, the mortality is about 75 per cent.

So far as the treatment is concerned, there are apparently two classes of cases, the infective and the non-infective. In the latter, the appropriate treatment is rest in bed during the acute stage, heart tonics while there is cardiac insufficiency, and when the acute symptoms have yielded, baths and resistant exercises. Perhaps at the last careful massage is advisable; certainly passive movements. Where there is doubt as to the nature of the disease, or a suspicion of infection, the blood should be examined, cultures being made from time to time, in order to establish or disprove the matter of infection. If the latter is certain, appropriate treatment should be instituted to combat the poison. At the moment, sera appear to offer a possible means to this end; inoculation of vaccines is also reported and has had good results occasionally. After cultures of the blood have been taken, serum corresponding to the nature of the infection can be administered. Treatment, to be logical, should always recognize the distinction between the infective and the non-infective forms of thrombosis.

Chapter XI.

MYOCARDIAL DISEASES.

The complaint of Bertin, the French internist, in 1821, that myocardial inflammation was wrapped in "a melancholy obscurity," seemed to furnish the stimulus for a long series of investigations which in the end have borne excellent fruit.

For, a hundred years ago there were no real distinctions between the many morbid changes that take place in the myocardium. So that when Laennec discovered fatty degeneration of the heart, the first positive advance was made in myocardial pathology.

But another quarter of a century elapsed before fatty degeneration was distinguished clinically from the fatty deposition of the fat heart. It was Stokes who traced the cause of this degeneration to infective diseases, notably typhus and typhoid fevers. Hayem confirmed Stokes' discovery by microscopic examinations. Meanwhile, in 1856, Ricord found that syphilis also attacked the musculature of the heart. Then Da Costa of Philadelphia, while studying the hearts of soldiers that had survived the campaigns of our Civil War, discovered his "irritable heart," the result of muscle strain, and reported his findings to the U. S. Sanitary Commission in 1867.

Up to this period our attention had been mainly riveted on the muscular tissue of the heart as the *fons et origo malis,* but Bristowe found that the interstitial tissue might also be involved. Soon afer, diphtheria and scarlet fever were proved to cause acute myocarditis, and then Huchard traced the source of interstitial inflammation to the coronary vessels. Finally, von Leyden of Berlin, in 1878-9, gave us the most complete clinical picture, up to that date, of the several varieties of the fat heart. These contributions have removed much of the obscurity that Bertin complained of in his quaint language.

As a result, we can now say that all toxemias, acute or chronic, some dyscrasias and hyperemias, if long continued or severe, and other conditions to be mentioned later, produce definite morbid changes in the heart walls, evanescent or permanent, as the case may be. It has been more difficult, of course, to connect characteristic clinical phenomena with each particular change, but even this has been done with such a degree of definiteness that, though important pathognomonic signs are still absent, we can nevertheless picture in our minds the several myocardial affections and their stages.

But to the profession at large these matters are not so well defined. Their attention has been directed mainly towards diseases of the endocardium or pericardium. Indeed, many of us have been keen to establish the precise location and degree of some endocardial lesion, to the comparative neglect of the myocardial, whose condition was really of greater importance.

The word myocardium, referring as it does to muscle tissue as the preponderating element in the heart walls, is to be understood here as a synonym for the heart walls themselves (Fig. 69). But in the word myocarditis we have to face the fact that its use has led to much misapprehension. Some still use it indiscriminately, or as the equivalent of myocardial disease in general, while to the majority it means myocardial inflammation, and therefore does not include the most common of all myocardial affections, degenerative changes.

About four years ago I proposed a new classification, to include all forms of myocardial disease. It was as follows:

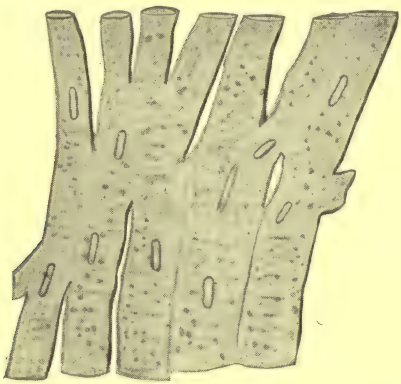

Fig. 69—Normal heart muscle, x 250.

1. Acute parenchymatous myocarditis.
2. Acute diffuse myocarditis, including the tuberculous, syphilitic, and suppurative forms.
3. Chronic myocarditis, including all of the diffuse inflammatory changes mentioned.
4. The fat heart.
5. The fatty heart.
6. Hypertrophies, whether due to severe exercise, vascular diseases, blood disorders, neurotic disturbances, possibly pregnancy.

To which I have added:

7. Atrophies.

While this book has been going through the press my attention

has been called to the so-called rheumatic deposits in the heart which Aschoff discovered in 1904 (*Verhandl. d. Deutschen Patholog. Gesellschaft,* Jena, 1904, II, 46 *et. seq.*)

Recently, Fraenkel has (*Beit. z. path. Anat.,* 1912, III. 597) examined twenty cases with a definite history of rheumatism, and found the nodules in all but three. According to him they are developed from the periadventitial tissues of the vessels, are microscopic in size, and consist of nodules made up of large cells with one or more irregular or polymorphous cells, and often but not always of small or large lymphocytes, as well as ordinary polymorphonuclear lymphocytes. Other changes than the above that have been noted have been hyaline degeneration of the muscle fibres of the heart, and also calcification of them. A. M. Pappenheimer, (*Proceedings N. Y. Path. Soc.,* 1910-11, N. S., X. 129), in describing a case that came under his observation has given the literature of the subject.

Neither dilatation nor spasm is included in my table. They are incidents which occur at times in any of the varieties mentioned and cannot therefore be considered separately.

Fig. 70—Acute parenchymatous change, x 250.

Some such classification as the above is essential for a clear conception of myocardial diseases, though many of the pathological phenomena here given are apt to be interconnected in any one instance. In fact, this complicated inter-relation has led some internists to emphasize the clinical aspects of these affections at the expense of the pathological. Now, it is true, in a general way, so far as symptoms and treatment are concerned at least, that chronic myocardial insufficiency is sometimes synonymous with myocardial abnormality or disease; but it is not so always. For example, the insufficiencies of the syphilitic heart and fat heart differ widely from

others of the myocardial group, so that they need to be classed separately, while the more we study these subjects the greater will be the variations discovered. Hence the importance of considering the affections apart.

By those who are familiar with ordinary pathological views as to the origin of acute affections in general, it will be admitted that parenchymatous changes in the cell elements of a part are such regular precursors of inflammatory conditions that they are to be considered as early symptoms. And so a parenchymatous myocarditis may be but the first indication of the diffuse form, even though the character of the parenchymatous change is not fully understood.

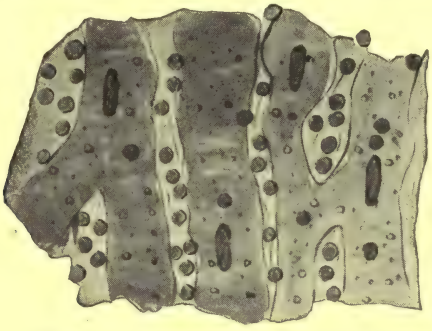

FIG. 71—Acute myocarditis, x 315.

However, it is to be presumed, from what we know of similar processes elsewhere, that parenchymatous changes will not only produce fatty degeneration of the muscle cells, but necrosis as well, leading to dilatation and perhaps even to rupture of the organ.

Taking up another feature of myocardial disease, I will say that we have not duly appreciated its frequency. The statistics of Dr. Harlow Brooks[1] of New York illustrate this point. Of 457 cases taken from his post-mortem examinations, where death was credited to cardiac disease, in 330 the heart walls were diseased sufficiently to cause death, while in many others heart wall disease contributed to the fatal ending; and yet in a considerable number the myocardial disease was not recognized during life, though, of course, much might have been done to arrest or control it. Now 126 of these deaths, or 38 per cent., were due to uncomplicated degenerative disease of the heart walls.

My clinical records point to the probability that in 45 per cent. of my deaths in heart cases, degenerative changes in the heart were

[1] Brooks, *New York Medical Journal*, 1907, LXXXV. 256.

the chief disposing causes of death; though uremic poisoning or other conditions may at times have been the actual causes.

Roemer of Tuebingen, while at Liebermeister's clinic, during a period of twenty years had a record of 44 per cent. of myopathic heart failures where there were no valvular symptoms, so that our clinical figures tally closely; while Brooks' figures, if applied to all his cases of myocardial disease, complicated and uncomplicated, might well corroborate mine.

Romberg has found chronic cardiac insufficiency (by which he means chronic myocardial disease) the most common of heart affections. At least, between the ages of 40 and 65 in men, and in women at a little later period, he found it more common than valvular disease. But I will go further.

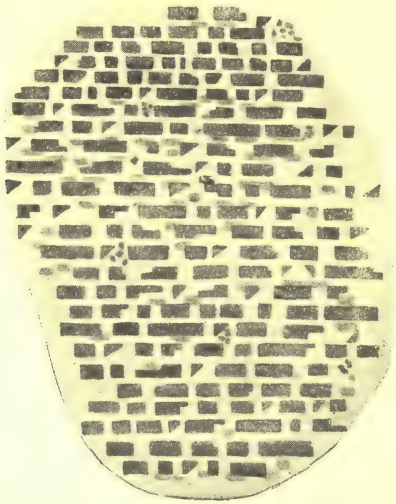

Fig. 72—Segmentation and fragmentation, x 110.

If to these chronic forms we add all the acute myocardial affections associated with infections, dyscrasias, anemias, intoxications, etc., we can well believe that myocardial disease is more frequent than valvular disease. Other cardiac diseases, however, than the valvular and myocardial are very rare, while valvular disease is tolerably sure to produce more or less myocardial disease; from which it is easy to see that the latter is the most common form of heart disease, and probably more so than all other forms combined. Schott (*New York Medical Journal*, 1909, LXXXIX. 65) says that diseases of the myocardium, in comparison with those of the endocardium, are as five to two.

Taking up now the morbid changes of the tissues, parenchymatous alteration is the earliest of all. In diphtheria, for example, it may occur within the first few days, or at any time up to several weeks after the fever has subsided. Acute parenchymatous change is characterized, pathologically, by the occurrence of granules in the muscle cells, and the partial disappearance of the cross-striations. This early parenchymatous change is probably analogous to the "cloudy swelling" seen in the uriniferous tubules at the onset of an acute nephritis (Fig. 70). It antedates, I think, every other sign. Though diphtheria appears to be largely responsible for myocardial disease (Schmalz found it in 16 per cent. of his diphtheria deaths), it also occurs in rheumatism, with or without the articular signs;

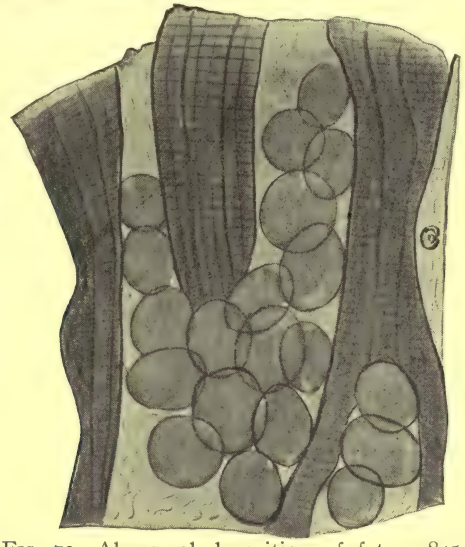

Fig. 73—Abnormal deposition of fat, x 845.

in tuberculosis, tonsilitis, measles, lobar pneumonia, erysipelas, epidemic influenza, septic processes, gonorrhea, and probably in all severe infections and intoxications. The parenchymatous change may be localized or general, usually the latter. Seen with the naked eye the muscle tissue is lighter colored than normal, while the cut surface is lack-luster in appearance, swollen, and moist. Microscopically the muscle cells are enlarged, less translucent than usual, and studded with minute particles derived from the protoplasm of the cells, and albuminoid in character, so far as microchemical tests show. In advanced cases the nuclei are similarly transformed. According to our present ideas, these changes are due either to the toxins of the above-mentioned diseases or the continued fever. In

alcoholism a similar condition has been noted. The mechanical effect of this change is naturally to limit the contractile powers of the muscle and cause cardiac debility sooner or later; so that the pulse may be increased in frequency in order to maintain the equilibrium of the circulation.

Now, inasmuch as we cannot dissociate these parenchymatous changes, clinically, from those of acute myocarditis that is a somewhat later event in the pathological chain, it will be convenient to describe the latter here. It presents a different microscopical picture. Here the muscle bundles are not only swollen with serum, filled with granules, and opaque, but the muscle striations may be nearly obliterated. Segmentation and fragmentation are said to occur occasionally.

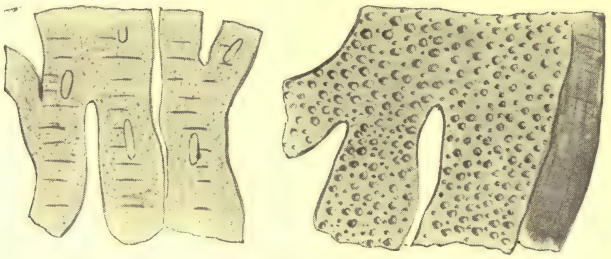

FIG. 74—Fatty degeneration of cardiac muscle. *a*, beginning changes; *b*, complete degeneration, x 250.

But we must not treat these conditions too seriously. They may be ante-mortem or post-mortem phenomena (Fig. 72). In all cases, however, the intermuscular tissue is swollen and infiltrated with blood globules and the proliferating cells of the interstitial tissues and vascular sheaths.

What we are pleased to call acute myocarditis is therefore a diffuse inflammation in which the muscular tissue is only one of several elements involved. It may be localized or not. Occasionally the process may go on to suppuration. Usually the abscess is embolic and multiple, sometimes due to infective endocarditis, to pyemia, or some other infection. The abscess is usually fatal, discharging into one of the cavities of the heart, or into the pericardium, causing aneurysm, pyocardium, or even rupture of the organ. But the acute form may pass over into the chronic. For example, an acute tuberculosis of the heart walls, which, though rare, does exist, may go over into the chronic form; and so in syphilis and probably in gout, and some other affections. In fact, there is no doubt that in all of these instances the disease which at first originated in the

larger cardiac vessels or their perivascular sheaths extends to the interstitial tissues along the lines of the smaller vessels. Even the elastic tissue may be involved, and it is a very important factor in the heart's action, expanding and contracting, and so aiding the muscular tissue.

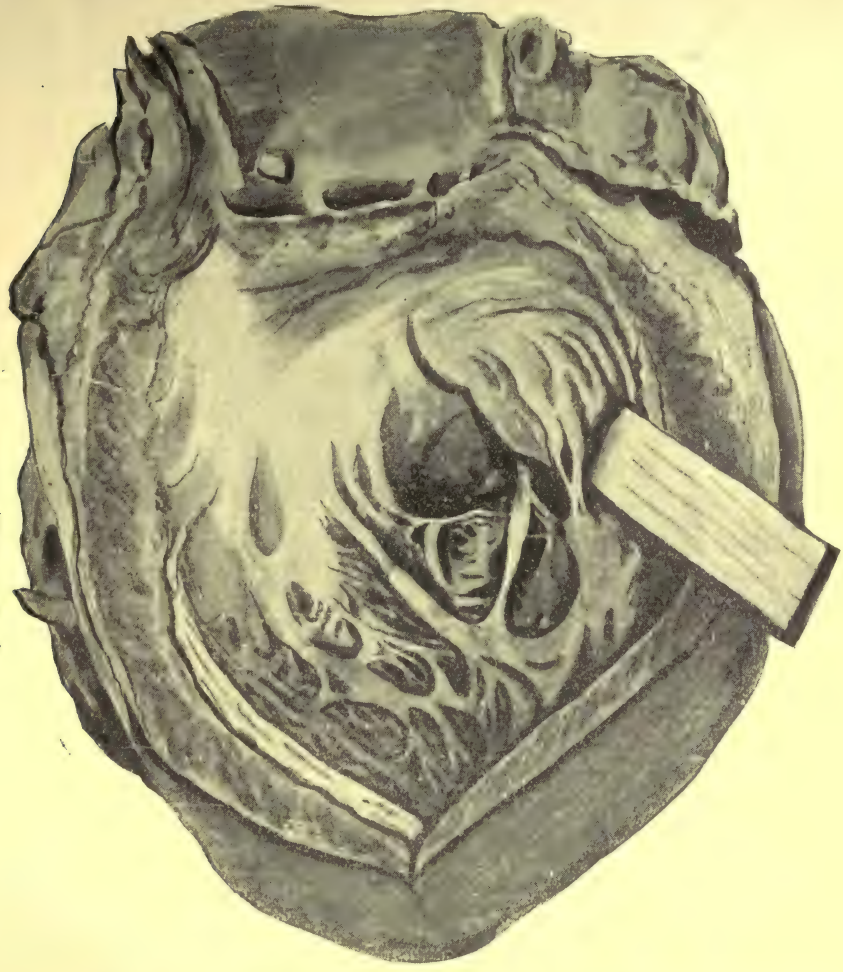

FIG. 75—Fatty degeneration of heart, with thickened aortic leaflets and mitral stenosis (wooden wedge in button-hole opening); about one-half size.

Among other causes of cardiac hypertrophy apart from severe exercise are arteriosclerosis, congenitally small vessels, scoliosis, Graves' disease, and the neurotic heart of hysteria. In arterio-

sclerosis of the coronary arteries, the sclerotic process starts from the vessels or their sheaths, and then extends along their subdivisions throughout the heart substance, increasing its thickness and density.

If the congenitally small vessels described by Virchow are present, the muscular tissue of the heart may hypertrophy in order to propel the proper amount of blood through the narrowed arterial channels. In scoliosis, as is now well established, the heart walls hypertrophy because the movement of blood through the heart chambers is restricted by the narrowing of the bony framework of the thorax, and some pressure must be brought to bear on the right or left ventricle, as it may be, in order to maintain the circulatory equilibrium. In Graves' disease and in the hysterical heart the increased activity of the organ, due to the neurotic condition, increases the heart's rapidity and, if continued, eventually causes muscular hypertrophy.

The terms fat heart and fatty heart imply some semblance of intimate structure. It is true they are closely allied, pathologically speaking, but in uncomplicated cases there is little clinical resemblance between them, as to natural history, diagnosis, and prognosis. In the fat heart, as we all know, there is a deposition of fat between the muscle fibers or bundles (Fig. 73), while the fatty heart is descriptive of a fatty degeneration of the muscle fibers themselves (Fig. 74).

In the fat heart there will not, as a rule, be any interference with the function of the muscle, though in advanced cases the fat may of itself interfere with the motility of the fibers by external pressure; while in fatty degeneration the protoplasm of the muscle itself has undergone a change that necessarily restricts its energy and may eventually destroy its vitality in whole or in part. There is no doubt, however, that the fat heart tends to degeneration; that is, it is apt to evoke in the muscle against which it presses, degenerative changes; but a fatty heart never becomes a fat heart.

The fat heart and corpulence are so closely allied that we cannot discuss one without the other. Quain, who made a special study of this subject, claimed that patients with fat hearts were invariably corpulent. I should modify his statement by saying that patients with fat hearts are generally corpulent. There is certainly a pretty constant connection between the two. And yet if a corpulent person is reduced by treatment the fat about the heart is apt to remain long after the external evidences of fat deposition have yielded.

On the other hand, beer drinkers and gluttons often put on

FIG. 76—Arteriosclerosis of the abdominal aorta, reduced to about one-third size.

bodily fat which their hearts do not share. With these exceptions, corpulence and the fat heart are practically coincident.

The fat heart may be said to occur at any age, but is rare in advanced life. For as the atrophic changes of senility set in, the deposits of fat usually disappear at the same time.

Von Leyden has claimed three varieties of the fat heart: (1) When there is a deposit of fat in the heart walls without organic

Fig. 77—Bramwell's "withered apple" heart; natural size.

affection of the muscle tissues. (2) A severe form associated with organic muscle disease. (3) A form complicated with disease of the aorta and coronary vessels, perhaps with kidney implication.

I am in doubt as to this third variety. As seen in Fig. 76, disease of the aorta is apt to be associated with the dilated heart of fatty degeneration, not fatty deposition. But there can be little doubt as to the occurrence of the first two varieties. Fatty degeneration,

however, is more common than the fat heart. Just as this moment the evidence appears to favor the idea that the oily particles come from without, though it is more natural to believe that they come from within the muscle cells and are sequels to the albuminoid changes of acute myocarditis, especially as they are found in the very class of cases that suffer from acute myocarditis, as in the eruptive fevers, diphtheria, acute rheumatism, lobar pneumonia, influenza, tonsilitis, cellulitis, enteritis, or any acute attack of toxemia; also in chronic intoxications, such as lead poisoning or uremia.

But fatty degeneration (Fig. 74) is also found in wasting diseases like carcinoma and paralysis, in prolonged fevers, after mechanical injuries or surgical operations, severe depression of the system, in pregnancy, and in senility. It is important to recognize these facts in order that we may be prepared for a sudden crisis. For when, in the affections named—especially in the toxemias due to diphtheria, in eruptive diseases, cellulitis, suppuration after operations—sudden death occurs, the probability is that the mischief is due to fatty degeneration of the organ, and the cause a toxemia rather than an embolism, heart clot, or cardiac paralysis, which in times past were terms used to explain these accidents. In fact we may go so far as to say that diseases of the heart substance in which fatty degeneration plays an important part are the chief causes of death in heart disease.

Predisposing if not governing causes of fatty degeneration, however, are cardiac hypertrophy, fibroid disease, the fat heart, endo- and pericardial diseases, including tuberculosis, new growths, and arterial disease; overexertion, overexcitement, abuse in eating or drinking, very likely anemia. It is unfortunate that coronary arteriosclerosis has been regarded by some as the main predisposing factor in fatty degeneration. Evidence does not sustain this view. Coronary arteriosclerosis is, in my opinion, one of the rarer causes of fatty degeneration. While it may occur at any age, the grosser forms are certainly associated with other degenerative changes that are the concomitants of senility. It is certainly more common in advanced age than middle life. So that being both the most dangerous form of heart disease and comparatively frequent, the importance of an early and correct diagnosis is apparent.

Unfortunately, its diagnostic features are not strongly marked, and in mild cases may be absent. In fact, there may not be any appreciable dilatation, and this is the one sign of paramount importance.

I have little to say about cardiac syphilis. It has been known to

exist for more than fifty years, but my experience has told me that the danger of it has not been appreciated. One reason is that it usually occurs very late after the initial lesion, even 8 to 10 years, when the patient has begun to forget that he ever had the disease; or if he has had it, has thought it cured. It should also be remem-

Fig. 78—Callosity at apex of heart in a case of purpura hæmorrhagica, about one-half actual size.

bered that cardiac syphilis may be hereditary. Some years ago I found from my clinical records that syphilis positively existed in five per cent. of my heart cases and probably in five per cent more. I hold to this position still. Certainly there is a class of heart cases with a previous syphilitic history, or perhaps with locomotor ataxia.

or some other affection of the central nerve system, where treatment will be ineffectual without the use of mercury or the iodides.

Atrophy of the heart occurs, but is comparatively rare. It may be a family peculiarity, or due to arrest of development, starvation, or some wasting disease, such as cancer or phthisis, or simply old age, when it is to be regarded as physiological. The atrophic heart has been known in adults to weigh only about three ounces, in fact a little less. These hearts in uncomplicated cases have a dark reddish-brown color and are in the condition that has been called "brown atrophy." The muscle fibers have yellow-brown pigment about the nuclei. Sometimes the organ is so shrunken that its vessels are thrown out into relief, "the withered apple heart" (Fig. 77). But there are no very distinctive physical signs of it during life. Of course, in uncomplicated instances, the heart's area will be smaller than usual. Otherwise the signs that have been described, such as faint turns, singing in the ears, palpitation, irregular pulse, precordial distress, and confusion of mind, will be suggestive, in conjunction with the diminished area, but they cannot be called pathognomonic. However, any one of us may be called in to see such a case, and the diagnosis will be made with reasonable certainty, if the heart is anatomically small in old age, or in any of the affections mentioned, especially if the pulse is weak and irregular, and there are fainting turns with cardiac distress. Such a heart came under my observation at a meeting of the New York Pathological Society, some years ago. It was taken from the person of a young man who died of sarcoma in the practice of Dr. R. E. Van Gieson. The heart weighed less than five ounces. In Bramwell's female cancer patient, the heart weighed only two ounces, twelve drachms, and ten grains. Fig. 77 is a life-size copy of Bramwell's case. According to Gibson, Quain knew of a more remarkable instance in a girl of fourteen, whose heart weighed only one ounce, fourteen drachms.

Fig. 78 is the heart of a female patient of mine who died at St. Luke's Hospital of purpura hæmorrhagica. The subserous hemorrhages are seen beneath the endocardium. There were similar effusions beneath the submucous tissues of the intestine and uterus. She had been under my care previously for a double lobar pneumonia, and had recovered without leaving any permanent fibrous adhesions in the pleural cavity, so that the idea then prevailing that fibrous pleurisy is never recovered from was disproved. At the post-mortem was found (Fig. 78) a cardiac callosity, or invading fibrosis, which, as no other explantaion offered, may perhaps have resulted from a previous pericarditis.

In Fig. 79 we see how an invading fibrosis can squeeze the muscular tissue.

Another method by which cardiac fibrosis develops is shown in Fig. 80. Supposing that in aortic endocarditis, as in Fig. 75, a particle is dislodged and finally arrested in a coronary artery; an infarct (white) may result. For the coronary vessels, though not terminal as in the brain, have really very slight capillary connections, and the plugging of an arterial branch may cut off from the neighboring territory so much blood that it is practically starved to death. As a consequence, the muscle tissue involved (with such vessels as exist) atrophy, and the whole area is supplanted by fibrous material. The cardiac walls are then correspondingly weakened, and lose a portion of the tissue which regulates the contraction and expansion of the organ.

The life of cardiac hypertrophy consists in a gradual thickening of the heart's tissues, with dilatation and enlargement of the muscle cells; perhaps an increase in their number.

During this period, which may be called the first stage, the pulse

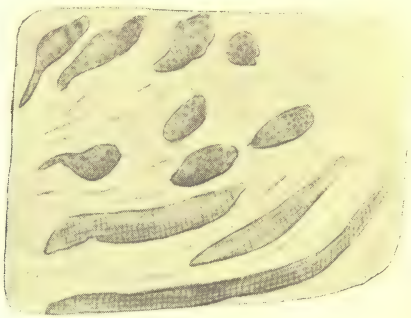

Fig. 79—Fibroid invasion of the myocardium, x 210.

is irregular, but its strength increases, the accentuation of the second pulmonary sound at the base indicating that the right ventricle has become enlarged. This sage is completed when the hypertrophy has been sufficient to propel the blood column with the required amount of force.

The second stage is marked by a return of the action of the heart and pulse to their normal conditions. This stage may continue indefinitely, so far as we know.

If, however, the dilatation increases, as it may from various causes, we then enter upon a period of failing compensation. This is the third stage. But now a sudden further dilatation will mean

heart failure, and, if no relief is given, immediate death. Loss of rest, excitement, insufficient nourishment, unusual exposure, sudden strain, may, any one of them, cause sudden dilatation of the heart and death. And yet we should remember that cardiac hypertrophy itself is in all instances a benign process, devised by nature to relieve a worse condition. For in all these cases, even in the hypertrophied heart of an athlete, the muscular tissue has doubtless been reinforced in order to restore the tone of the organ, in recompense for a strain, or, in other words, a laceration.

The diagnosis should present little difficulty. Except in very stout persons or women with pendulous breasts, the outline of the heart can usually be made out with sufficient accuracy. Even if it cannot well be defined, the apex beat can be located by the stethoscope.

Cardiac hypertrophy therefore indicates the existence of some systemic primary defect or disease, that governs the prognosis; unless there happens to be some intercurrent incident or accident to provoke heart failure. Cardiac hypertrophy in nephritis should not,

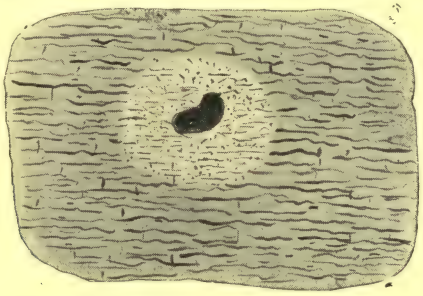

Fig. 80—Cardiac fibrosis and the white infarct, x 67.

of course, be taken too seriously; the nephritis is the chief danger. But the athlete's heart is more liable to suffer from excitement or sudden strain than the normal heart, so that he should be warned, in order to guard himself against sudden dilatation. In mild cases, however, the hypertrophy may entirely disappear.

A patient of mine out hunting suddenly realized that he had strained his heart in attempting to roll over a heavy log to build his camp. He was a physician, and was fully alive to the fact that his heart had been permanently disabled. Subsequently, under a sudden dilatation from a cause incidental to the life of any busy practitioner, he was taken off without warning. The fatal issue in his case could hardly have been avoided, so long as he discharged the duties of his very responsible and arduous position.

In my experience, also, a patient may suffer for years from a severe strain, and ultimately recover from it. A male patient of mine, twenty-eight years of age, has entirely recovered from a severe cardiac strain which happened eight years ago.

In any case of suspected dilatation, it is extremely important to determine how much hypertrophy is present, certainly to the extent of noting the altered location of the apex beat or sound. Dilatation is, as we all know, characterized by a soft, weak, and irregular pulse, which is usually quite rapid, unless the nervous system is depressed, or there is renal toxemia. The dilatation may be more or less extensive. But, clinically speaking, if the left margin of the heart does not extend beyond the nipple, it is not an alarming sign; if much beyond that point, the prognosis is unfavorable. In mild cases, however, the dilatation (as shown by the apex beat) will not reach to the nipple.

If, now, in the course of or after typhoid fever, diphtheria, or any of the acute or chronic toxemias mentioned, the pulse becomes weak and irregular, no matter what its rapidity, attention should be directed to the probability of some myocardial affection, and if there is precordial distress, or cyanosis, dyspnea, some edema, or, in extreme cases, anginoid attacks, a diagnosis of acute dilatation can be made with a reasonable degree of probability, which will amount to a certainty, if it is found that the heart's dimensions have suddenly been enlarged.

In such instances we should give the heart, as far as practicable, relief by rest in bed; for the horizontal position relieves the weakened heart, the diminished number of cardiac contractions accomplishing the desired result as well as a greater number when the body is vertical. If the parenchymatous change is associated with a pyrexia, sponging of the surface with alcohol and water will reduce the temperature, while there may also be a field for an antipyretic drug, such as aconite, in small doses.

In apyretic cases, as in the various forms of chronic cardiac disease, it is more logical to relieve the peripheral pressure by warmth to the extremities, or by the use of nitrites or nitrates, than to stimulate the heart. For whatever temporary gain may be obtained for the tired heart by a cardiac stimulant, cardiac activity will diminish as soon as the physiological effects of the drug have passed away.

We should also hesitate to give drugs merely for the purpose of lowering or increasing the pulse rate, because the rate may be adapted to the pathological condition. Through the influence of the

nerve centers, central or peripheral, there is always a compensatory action of the heart, adapting its speed or pressure, more or less certainly, to varying conditions. Of course, when the acute symptoms have received proper attention, no time should be lost in endeavoring to eliminate the toxins (if there has been toxemia), by such means as are calculated to remove or nullify the primary cause of the dilatation.

Von Leyden's views as to his first two varieties of the fat heart are valuable. In the first of them there may be shortness of breath, asthmatic attacks, and a rapid pulse, even when the heart muscle is not involved, caused by the extraordinary efforts made in the movements of the obese, owing to mechanical interference of the various fat deposits with respiration and circulation. Such persons seldom live to old age and succumb very easily to intercurrent infective diseases. This form, however, is curable under appropriate treatment, such as baths, exercises, gymnastics, and diet. But under certain circumstances, such as psychic disturbance, overeating or drinking, or training, it may pass into the second form.

In his second form[2] the prognosis and treatment are different. The prominent symptom is heart weakness, with precordial distress at any time, and orthopnea, while the blood is apt to stagnate in the veins, and there is cough and bloody expectoration, swelling of the liver and atrophy. The heart dilates, but the auscultatory sounds vary. The normal sounds are weaker, however, the apex beat less easily recognized; or there is a splitting of the sounds, or sometimes a systolic murmur at the apex due to muscular weakness at the mitral. The second sound above the aorta is usually stronger than the pulmonary. The action is also arhythmic, showing a defective action of the left ventricle. The pulse is small and irregular, but may be slowed or hastened.

When arteriosclerosis is detected in the peripheral vessels; or the contracted kidney by urinary analysis, we can realize that a fat heart already weak is meeting increased resistance which must tend to dilatation and heart failure.

Passing over its etiology, the diagnosis of the fat heart is comparatively easy, though always a matter of inference. It cannot be determined by physical signs. However, as Quain says, in stout people when the pulse is small and weak, the first sound feeble, the impulse weak, and the heart's area enlarged, it may be judged that there is a fat heart, with a fair degree of certainty.

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[2] *Russ. Med. Rundschau*, 1907, 276.

So far as treatment is concerned, it does not present much difficulty, provided the patient takes his condition seriously, and will faithfully carry out some rational plan. A physician, seventy-six years of age, weighing 377 pounds, and unable to walk without assistance, I reduced over forty pounds in a few months, with very little difficulty, relieving his rheumatism and chronic eczema, so that he was able to resume, to some extent, his outside practice. He took several courses of the Nauheim treatment, and adhered to the rigid dietary I laid down.

Some, however, women especially, are quite willing to take any prescribed course, provided only their dietary is not restricted, and consequently treatment with them is a failure.

My dietary avoids fat meat, bread that contains much starch, and coffee, but often includes the use of sodium bicarbonate, advocated by Trousseau. I have found that my plan will reduce at the rate of from four to ten pounds a month, a rate that is sufficiently fast. Nor does it preclude tonics, wine, or other stimulants in moderation. As a matter of fact I have not had occasion to us alcoholics. My dietary is as follows:

8 A.M. Breakfast: three ounces lean meat without fat; one ounce gluten or whole-wheat bread, toasted; a cup of postum with milk, or lemonade.

10.30 A.M. A cup of beef-tea.

1 P.M. Five ounces of lean meat or fish; salad with French dressing.

3.30 P.M. Eight ounces of milk and Vichy water (half and half).

6 P.M. Three ounces of rare meat or fish, with pickles or salad; one ounce graham bread, toasted; stewed fruit.

8.30 P.M. Eight ounces of milk and Vichy water (half and half).

Vichy or plain water can be taken as desired during the day. This is not a severe dietary, as compared with some that have been recommended.

The general principles under which I practise reduction are restriction in the amount of sugar, starches, and fats. Enough water should be taken to cause the normal amount of urea to be excreted. Oxidation should be increased by resistance exercises and by carbonated baths, which stimulate the skin and so improve the capillary circulation. Tea and coffee appear to restrict oxidation, and should for that reason, if for no other, be prohibited, or taken in extreme moderation.

Acid fruits and drinks should be taken very sparingly. An ex-

cess of them produces indigestion. They are better cooked. Sometimes all fruits should be forbidden.

In general, however, small fresh fruits may be taken with discretion, in their season. Sometimes the amount of both liquids and solids has to be much reduced. Laxatives should be taken so as to produce two full fecal movements daily; and stomachics also, if indigestion is acute. The patient should also take regular walks on level ground, of a given distance, and the distance should be gradually increased. Carbonated baths and resistance exercises are also valuable as auxiliaries in the treatment. But the vitality of the patient should never be reduced. On the contrary, it should continuously increase.

Stubborn cases may require the use of the hot box, or some other form of hot air apparatus.

With these methods it is practicable to reduce the weight without detriment to the general health.

In any case, however, before instituting treatment we should determine whether the fat heart belongs in Leyden's first or second class, and order the treatment accordingly. Obesity after middle life should be handled with extreme caution. A few years ago a contractor past middle life consulted me for heart weakness associated with obesity. He was leading an active out-of-door life. I put him on a restricted dietary, directed him to lead a less active life, and advised him of danger, in case he neglected my warnings He did not visit me again, and within two weeks fell dead, dancing at a ball.

This man was to outward appearance in robust health, notwithstanding his obesity. The lesson from such a case is obvious. As old age approaches the danger to life advances progressively in obesity, so that after middle life, as I have said, we should always be cautious in the treatment of these patients.

Romberg maintains that coronary disease is the most frequent cause of fatty degeneration, and that the diagnosis of coronary disease as its cause is certain whenever there is angina pectoris or cardiac asthma with sclerosis of the aorta, in a person over forty years of age. In this country most attacks of angina pectoris are held to be of nonorganic origin. This is my view. Sclerosis of the aorta cannot be positively determined during life, and cardiac asthma is common to heart failure in any heart affection.

Some years ago I divided fatty degeneration into three stages, making the pathological changes conform to the clinical signs, and gave instances of each, as proved by my post-mortem examinations.[8]

[8] Diseases of the Heart, etc., N. Y., 1905.

According, however, to more recent views, the first fatty changes I then described are to a certain extent coincident with changes of an inflammatory nature, as in typhoid, and subsequent investigation may show that the changes I then described are secondary to more acute manifestations, so that my first stage of fatty degeneraion was coincident with inflammation of the heart walls. This is a matter that requires further study. In my first stage, (the period of acute myocarditis of some) the duration is not long (as in the eruptive fevers, typhoid, or other acute toxemias), and the symptoms, such as a weak and intermittent pulse, feeble beat, or distant sound at the apex, with faint tones, and more or less dilatation, precordial pain and distress, will mark the first stage.

These signs I noted as early as 1879 in a fatal case of yellow fever that occurred at the Presbyterian Hospital, when I was pathologist to that institution. Of this I have my post-mortem and laboratory notes. In this stage the heart may stop; not, however, from the fatty degeneration observed by Hayem in the sudden death of typhoid fever, but from the toxemia, causing defective innervation. For the central nerve system regulates the action of the heart, though it may have nothing to do with its rhythmic activity. To put it physiologically, the contraction and relaxation which characterize the rhythmic action of the heart are due to the interaction of an alternating kind between the sodium, potassium, and calcium ions, and the contractile substance of the heart muscle.

In the second stage, the signs of a dilated heart are more pronounced. The left ventricle may reach to the left nipple, or extend beyond it; the right ventricle an inch or more beyond the right border of the sternum. The apex may be in the fifth, sixth, or seventh space, and the apex beat feeble. The pulse will usually be soft, intermittent, and infrequent. There may be, and often is, a lack of harmony between the heart and pulse-beats. These manifestations of arrhythmia are apt to be seen in persons who have passed middle life, and are inclined to be stout, but are at the same time anemic. In these instances there is often a distinct whiteness about the face, which is a noteworthy sign. This is common among the upper classes, especially with those who lead a rather inactive life.

The apex beat will be located with difficulty, or not at all. There will be dyspnea and some precordial oppression, with occasional attacks of dizziness. Abdominal symptoms will always be in evidence; occasionally gastric crises alternating with anginoid attacks. Cyanosis will occur at times. Such patients will often be irritable

or whimsical, or nervous about trifles, and always concerned about their health. There will be a disinclination to undertake anything new, even to walk, and the gait may be uncertain. Occasionally, there will be hemorrhages, usually in the form of epistaxes. Edema of the face, hands, and feet will occur at times. Independent of valve murmurs due to obstruction, ther may be systolic murmurs from leakage at the mitral, and if there is hypertrophy of the right ventricle, there will be accentuation of the second pulmonary sound. If acute dilatation takes place there will be precordial distress, pain, and dizziness, or a faint feeling. And there will be sharp anginoid atacks, if the dilatation is very acute.

If there has been corpulence it will help in the diagnosis; also if there is dyspnea after exertion.

Other signs which I regard as very important are—that the apex beat is not to be detected in persons of otherwise normal build; also, that the patient must sleep high at night with several pillows, or resting on his arms.

The urine may contain a little albumin and often a little sugar. This stage may last from two or three, to ten or fifteen, or even twenty years, depending on the degree of the degeneration and the ability of the patient to care for himself intelligently.

In the third stage there is implication of other organs. For example, there may be attacks of cerebral thrombosis or embolism due to abnormal heart action; or thrombosis of peripheral veins, but the circulation may be reestablished, and the patient resume an otherwise tolerably active life. Or, from a variety of causes, perhaps from indigestion, there may come a fatal attack of heart failure. Or, in old people particularly, a low form of meningitis, usually basilar, will develop the Stokes-Adams group of symptoms, with possibly the so-called "heart block." And yet even here the symptoms may yield and life be continued for months or even years, as in one of my cases of the Stokes-Adams type. Finally rupture of the heart may occur. But it must be exceedingly rare. Nephritis is the common terminal affection.

In the first stage the patient should be treated by rest and an appropriate but rather restricted diet, and general tonics, among which strychnine should have some part. It should be given in small doses, say from 1-50 to 1-60 of a grain, for adults, and should not be administered continuously for more than two weeks at a time. This, I hold, is important.

In ordinary cases of fatty degeneration the patient, if anemic, should be put on iron; if tuberculous, on malt preparations and creosote or cod-liver oil; if arteriosclerotic, on the iodides; if rheumatic, on an antilithemic diet and antilithemic remedies, in conjunction with the hot-air treatment.

In the second stage the Nauheim system is appropriate in selected cases. In the third stage, where all hope of cure is at an end, the use of strophanthus and drugs of the digitalis group, the nitrites and nitrates, including glycerol nitrate and erythrol nitrate, camphor, and finally oxygen gas, have each their distinct fields of usefulness.

There is a tendency, however, to use adrenalin in these cases, so that the experiments of R. M. Pearce (Studies from the Bender Hygienic Laboratory, 1906, III. 51) should serve as a warning. Injecting 1-10 to 8-10 of a cubic cm. of this drug into a series of rabbits, sufficient to cause death sooner or later, the heart muscle showed more or less edema, and in one instance wide separation of muscle fibers. In another series, each rabbit heart was dilated, while there was granular and hyaline degeneration of the fibers, and some were necrotic. Adrenalin contracts the arterioles, and it is conceivable that the contractions of the coronary and other vessels produce temporary ischemia in the heart walls. In some of the rabbits there was a diffuse myocarditis. To be sure, adrenalin produces prompt physiological results when injected into a vein. But I have seen five minims of an intravenous 1-1000 solution, (1-200 grain) produce distressing symptoms in a patient, causing a blood pressure of nearly 200 in forty seconds, and a corresponding fall in from one to two minutes.

I have, however, been in the habit of employing the suprarenal extract in doses of one to three grains of Armour's powder, in capsules, three times a day, as a substitute for glycerol nitrate in the weak heart, and have found it useful, and have observed no bad effects from it. But in reference to the adrenalin chloride, we must realize that there is a lack of constancy in its action. I am prepared from my experience to say that the same amount given to the same person will not always have the same effect. These statements are confirmed in a measure by two blood-pressure charts of T. C. Janeway. (Clinical Study of Blood Pressure, 1904.) In one, ten minims of a 1-1000 adrenalin chloride solution were injected into the substance of the tissues. No effect was produced. In another case, twenty minims of the same strength were injected into the median basilic vein. In fourteen minutes the pulse had risen to 145 and the pressure to 225, but the latter had fallen at the end of thirty-three

minutes to 55, and, in fact, no pulse was palpable, when it was found necessary to inject two grains of caffeine sodium salicylate, strychniæ sulphate, etc., to restore pulse and pressure.

I recently saw, in consultation, a patient where, in my opinion, the fatal issue was hastened by adrenalin. Physicians should, therefore, be on their guard against the internal administration of this particular preparation; viz., the adrenalin chloride. Its prompt action in surgical shock should not blind us to the fact that the primary action is followed by a dangerous depression.

So far as prognosis is concerned, in myocardial diseases, we must assume from analogical reasoning, together with clinical experience, that parenchymatous changes, if not excessive, can disappear with convalescence, and yet, by analogy, we must equally well hold that they can lead up to acute diffuse myocarditis; and may terminate in fatty degeneration also. In very many cases, however, we know that the patients recover wholly; at least so far as we can determine by clinical tests.

The same process of reasoning leads us to believe that even in acute interstitial disease the fibroid deposits may at times disappear entirely. So in tuberculous myocarditis there may be a disappearance of all signs of the tubercles, as in tuberculous peritonitis. The same may be said of syphilis. Moreover, a moderate degree of fatty degeneration is present in almost every adult heart, and if the post-mortem examination displays the so-called "tiger's heart," where the striations of fatty degeneration show in places through the transparent endocardium, one would hardly call it abnormal.

Reverting for a moment to cardiac syphilis, Huchard has claimed that angina pectoris is an important sign. This has not been my experience. Herzog's three signs are light precordial pain up to angina, tolerably rapid cardiac enlargement in young people or persons of middle age, with aortic insufficiency. Whether these points have value, time only will determine. Certainly organic disease of the arch of the aorta, or any thoracic aneurysm, would point towards syphilis. But cerebral signs are also suggestive. I had some years ago under my care a physician of New York past middle life, who gave no subjective specific symptoms of syphilis, but had suffered from chronic cerebral pains, in connection with his cardiac disease. A thorough course of mercurials and iodides, together with Nauheim baths and exercises, put him on his feet, and he is now, or was until quite recently, actively at work. So that the prognosis is not altogether bad. If the diagnosis is made early and the proper treatment instituted, success may be expected. In fact, there is no

reason why a cure cannot be made. Even in advanced cases, where there is tabes dorsalis, for example, great improvement can sometimes be effected. Iodides and mercurials are the proper remedies, but mercury is the sheet anchor. Sometimes both of them should be given for months, with brief interruptions of a few weeks. My experience with salvarsan in these cases has been limited but satisfactory.

Apropos of the diagnosis of cardiac insufficiency as shown by the soft, rapid, and often intermittent pulse of myocardial disease in general, it is well to remember that there may be persons whose hearts functionate sufficiently well, until they are subjected to some special strain, physical, mental, or psychic. Then the insufficiency is apparent. Often a few steps about a doctor's office is all that is required. To determine the amount of this weakness various methods have been tried for more than a century. In fact, the determination of the functional capacity of the heart is a matter of very great importance, not only from the point of view of diagnosis, but for the purpose of controlling the effects of treatment. But the methods used have been open to so much criticism that internalists have usually given the subject a pretty wide berth. The first material advance was made by Spengler, in 1887, when he noted the effect on the pulse in typhoid fever convalescents after exercise and change of posture. Of all the methods proposed, three may be taken as examples of the most recent that have been published.

Katzenstein's, published in 1905, consists in compressing the femorals or iliacs below Poupart's ligament and noting the effect on the brachial artery. In healthy persons he has claimed an average rise of 5 to 15 mm. Hg. in from two to five minutes, as shown by the sphygmomanometer. In weak hearts, he has reported that the pressure either rose slowly or not at all, or fell, according to the grade of weakness present.

As to the accuracy of the method, reports have differed. In acute cases it is, of course, inapplicable. Compression of these vessels is not an easy task; an assistant is necessary to take the pulse. In ordinary private practice, a patient might very reasonably object to the procedure.

Herz (*Deutsch med. Woch.*, XXXI. 215) has proposed a more practical plan—counting the pulse long enough to determine its average rate per minute. He then makes the patient sit down and slowly flex and extend the right forearm, continuing the movement a minute, without contracting the muscles the muscles to any marked extent, the physician controlling the

flexion and extension, but not using any force. In the normal heart the rate is not affected, but in the weak heart it is slowed from five to twenty beats per minute.

The older method of Schapiro is another good one that should be practised more than it is. The pulse is taken first in the recumbent and then in the sitting position. In health, there is an increase of frequency when the individual has assumed the sitting posture (after lying down) of from three to ten beats per minute. In the weak heart, there is an absence of or a diminution of this frequency in changing from the recumbent to the sitting position. I have used this method for the past four years, with a fair degree of success.

We have no better methods available than those of Herz and Schapiro, and, inasmuch as they are simple, practical, and fairly reliable, we should use one or the other to test the functional capacity of the heart, certainly until something better presents itself.

Chapter XII.

MALIGNANT ENDOCARDITIS.

Malignant endocarditis is a very serious disease, often difficult of diagnosis, and usually fatal, even if all the resources of the physician have been called into play. Among pathologists it is believed to be a simple endocarditis which has become infected by poisonous germs. Itt hen assumes so grave a type that the word malignant is an apt prefix. How long it has been known under this name is dufficult to say; Bouillaud in 1835 found that it had been in use before his day. This great diagnostician described both a typhoid and a septic type, which latter he called gangrenous; he also recognized three varieties, the acute, subacute, and chronic. In 1889 the term malignant endocarditis appeared in Wilkes' and Moxon's Pathological Anatomy, apparently because in 1882 Goodhart of Guy's Hospital found a group of fatal cases associated with typhoid and scarlet fever.

Simple endocarditis, meaning the non-malignant variety, is not to be regarded as a fatal disease, except in so far as it produces death mechanically through embolism of some important organ, the brain or basal ganglia, for example. Even cerebral embolism may occur a number of times before there is a fatal issue, which only results after the embolism has cut off the blood current from important nerve centers.

Malignant endocarditis is so rare a disease that no one person can have much personal experience with it. Nor can anyone discuss it intelligently without having studied a large number of cases at first hand. I have had the unusual opportunity of studying one hundred cases in the record books of some New York hospitals, and the chief object of this paper is to present a brief analysis of what my examination has yielded. In so doing I take this opportunity of thanking the officials of the several hospitals for the privileges they have accorded me and the facts they have put at my disposal. It is needless to say that I have endeavored to be accurate in the brief analysis I have made. Ther was no selection of cases; they were tabulated in the precise order in which they occurred in the several hospital books.

With regard to the infecting agent, only those of the streptococcus, staphylococcus, pneumococcus, and gonococcus groups were

recorded, either as having been found in the living blood or in the body after death. This list is a limited one, if we consider that the meningococcus, the Bacillus coli, the Bacillus typhosus, the Klebs-Loeffler bacillus, the gas bacillus, and probably others have been charged with causing the infection. Apparently there are epidemics of the disease, as is implied by the fact that Billings in eleven out of fourteen cases found the pneumococcus as the infecting agent (78 per cent). Among the associated systemic diseases I found rheumatism in more than one-third of the cases, and suppurating foci, pneumonia, pleurisy, phthisis, syphilis, gonorrhea, influenza, tonsilitis, chronic peritonitis, chronic appendicitis, colitis, and nephritis in varying proportions.

Unfortunately for scientific nomenclature, the affix ulcerative came to be used for this form of endocarditis, though ulceration merely represents one of the regular events in the simple form. In fact, it has been claimed that infection of valves may occur without ulceration of their surfaces, just as in diphtheria we occasionally see a diphtheritic patch on an unabraded surface. Less objection is to be charged up against the words infective and infectious, and yet we have not thus far been able invariably to find the pathogenic germs, so that these terms constitute a begging of the question. In fact, in my one hundred cases the germ was found in only 33 per cent. The same objection applies to the word mycotic, which is sometimes used. In speaking of the septic form, we of course allude to those assigned to the streptococcus, the staphylococcus, and other pus-producing germs.

In this connection I should state that the cases of which I present a brief analysis were sometimes examples of mixed infection. Thus, in my one hundred cases, of which thirty-three showed specific germs in the blood or elsewhere, streptococci were found alone in eighteen; staphylococci occurred jointly with them in one; staphylococci occurred alone in five; pneumococci were associated with them in two; pneumococci occurred alone in six; and the gonococcus in one. It is clear, therefore, that the predominating germs found were of the pus-producing varieties.

In malignant endocarditis there is a poisoning by implantation in the inflammatory deposit of the valves; in adults chiefly those of the left heart, and in infants of the right heart. The poisoned emboli, together with aggregations of bacteria, are carried forward by the blood current into the greater circulation, infecting the entire system.

As to the frequency of malignant endocarditis in this city, in the

Babies' Hospital, in a total of 8,775 admissions during the term 1900-1911, the records do not show a single instance of the disease. This institution presumably limits its admissions to those under three years of age. In the New York Hospital and St. Luke's during a similar term, with admissions numbering 102,631, but one case had been recorded of a child under three years of age. So that under the age of three or four it must be extremely rare; in fact, it almost never occurs.

My statistics, based on 100 cases, are as follows: Under 14, 7 per cent.; between 20 and 30, 25 per cent.; between 30 and 40, 30 per cent.; between 40 and 50, 18 per cent.; between 50 and 60, 4 per cent.; over 60, 1 per cent.; age unknown, 15 per cent.

We are led to believe, therefore, that this is largely a disease of early middle life, occurring with greatest frequency between the ages of thirty and forty, almost never under four, and seldom before fourteen or after sixty. Unfortunately, however, it is not possible to verify this statement by any official data obtained by governmental inquiry. For we are confronted by the fact that malignant endocarditis is not mentioned in the international list of the causes of death furnished by the Department of Commerce and Labor, nor does it appear in our National Census tables, or in the reports of our State Department of Health or our City Department of Health. The following abstract of a letter written me by Dr. William H. Guilfoy, Registrar of Statistics in our City Board of Health, under date of February 11, 1911, illustrates the prevailing method by which the returns are made by physicians in our city. He says: "I am unable to furnish you with definite figures in regard to the number of deaths from malignant endocarditis, either acute, subacute, or chronic; and, by the way, I do not remember ever having seen a death ascribed to chronic malignant endocarditis. My impression is that a majority of the deaths which are classified under acute endocarditis are deaths from acute or subacute malignant endocarditis, which have been reported as either acute endocarditis or septic endocarditis or ulcerative endocarditis."

In the Manhattan State Hospital for the Insane, with a total of 58,042 patients treated during the ten-year term ending September 3, 1910, not one case of malignant (infective or infectious) endocarditis was registered as such on the books. On the other hand, in Bellevue Hospital, where between 1902 and 1908 there were a total of 204,690 patients, 222 were registered as infective (malignant) endocarditis, including both the acute and chronic forms.

My statistics were taken from the records of three of the great

semi-private general hospitals, wher I was able to get from each the number of patients admitted during the last ten-year term available, together with the number of cases of malignant endocarditis registered during that period. The results were as follows: New York Hospital, 50 cases treated, admissions 88,365; St. Luke's, 45 cases treated, admissions 14,266; Presbyterian, 118 cases treated, admissions 33,592; total, 213 cases treated, admissions 136,223, or one in 639 patients.

In Bellevue Hospital the proportion was 1 out of 922, during a six-year term, with 204,690 admissions.

As a rule, with exceptions in very rare cases, malignant endocarditis is accompanied by fever. In one there was a temperature as low as 98 deg. in a woman of 41, who died with symptoms of chronic peritonitis. She was not in the hospital more than one day, and was probably in a state of collapse when admitted. In the larger number of cases the temperature reached or exceeded 104 deg. F., while in 73 per cent. it varied between 103 and 106 deg.; in 14 per cent. it touched or passed 106 deg., while in 2 per cent. it exceeded 107 deg. High temperature, therefore, was the rule. In only 7 per cent. was the temperature under 102 deg. The pulse rate seemed to vary with the temperature.

While in my cases germs were only found in 33 per cent. (in one of the hospitals they were found in 42 per cent.), but sometimes not until after death.

Cultures should be made in every case of the disease, and if the early ones are negative they should be repeated at short intervals, as the micro-organisms may not be found until the examinations have been many times repeated.

Petechiæ were noted in 19 per cent. But they must be differentiated from the rose-colored spots of typhoid and from purpura hemorrhagica. In this connection it must be remembered that there is also a purpura rheumatica.

Hemorrhages were occasionally noted, but only in 5 per cent. Implication of the spleen was found in 8 per cent. Headache and diarrhea were frequent as might be expected. The leucocyte count varied from 6,650 to 36,000; it exceeded 20,000 in only 5 instances. In 21 out of 33 examinations it did not exceed 15,000 (63 per cent.). The hemoglobin content was low, most often under 60. Rheumatism was a concomitant in 37 per cent. Pneumonia came next in frequency (6 per cent.). Tuberculosis existed in 5 per cent.; pleurisy and syphilis each in 3 per cent.; gonorrhea in 2 per cent.; acute and subacute nephritis in 2 per cent.; cerebrospinal meningitis in 1 per

cent.; mammary abscess, suppurative arthritis, colitis, chancre with suppurative buboes, chronic appendicitis, gangrene of foot, and miscarriages each in 1 per cent.; total, 66 per cent.

Sometimes the diagnosis presents great difficulties, and has to be made by exclusion, for often the symptoms are variable and indefinite and lead to the confounding of the disease with tuberculosis, purpura, typhoid or malarial fevers. The subacute or chronic form will perhaps for a time be difficult to differentiate from typhoid. It is here, however, that blood culture comes to our aid, for by the discovery of the streptococcus or the staphylococcus, where the Widal, von Pirquet, and malarial tests are negative in the presence of an endocarditis, the diagnosis of the malignant form of the disease may be made with reasonable certainty.

But we cannot depend absolutely upon culture methods. They fail in a certain number of cases, and the germs may not be recovered during life. We must rely on a diminished leucocytosis and enlarged spleen, hemorrhages, a previous endocarditis, some displacement of the heart, precordial pain, headache, rapid pulse, a temperature that is continuously high, a tendency to septic symptoms, and often suppurating foci somewhere. And inasmuch as the pathological diagnosis may be inconclusive, we must learn not to wait for it, but make, if we can independently, a clinical diagnosis.

Basing our prognosis on the 100 hospital cases, we may conclude that in malignant endocarditis 85 per cent. of the patients die, 10 per cent. improve for a time—how long is uncertain—but that in 5 per cent. there is such a degree of improvement that it may be called recovery, so far as the malignant nature of the disease is concerned. While we cannot put it in the same class with pyemia, the disease is to be classed with the severest forms of constitutional infection.

The duration of the disease may be from a few days to several months. Billings has reported a case in which the patient lived more than two years. In my one hundred cases the number of weeks spent by patients in the hospital before the fatal issue from one to thirteen, but the majority succumbed before the end of the fifth week.

Antigonococcal serum was used in 1 case, other sera in 3 cases, and vaccines 5 times, of 100,000,000, 60,000,000, and 24,000,000 strengths. In no case was there recovery under these forms of treatment.

Case I was that of a young man 21 years of age, who entered the hospital with suppurating glands in both groins, due to a chancre. He also had rheumatism. His temperature varied from 97.8 deg.

to 99.8 deg. F. He remained in the hospital 13 days, when he was discharged "improved." Apparently his treatment was symptomatic.

Case II was that of a girl of 7, in whom the diagnosis was made by exclusion. The von Pirquet, Widal, and malarial tests were negative. Her temperature rose to 104 deg., and her case was diagnosticated as one of malignant endocarditis. She improved so that when she was discharged after 13 days in the hospital she was nearly well. The diagnosis was clinical.

Case III was that of a man 30 years of age, who came into the hospital with influenza, rheumatism, and enlarged spleen. He had petechiæ, and a leucocyte count of 10,400. His temperature rose to 102 deg. Sputum negative. After 4 days he was discharged in good condition.

Case IV was that of a young woman 18 years of age, who entered the hospital with pleuritic effusion and rheumatism. The examination of blood was at first negative, but afterward streptococci and staphylococci were found. Her temperature rose to 105 deg., but after 96 days in the hospital she was discharged improved. She was ill in all 9 months. The treatment was symptomatic.

Case V was that of a man 25 years of age, whose leucocyte count varied from 32,000 to 18,300, while the Widal test was negative. The diagnosis was subacute rheumatism. His temperature rose to 103 deg., but under symptomatic treatment it gradually fell, and he was discharged improved. He was 28 days in the hospital. How far this improvement was maintained is a matter of interest to us, for of these cases I have no further information.

As treatment by sera or vaccines was employed in only 9 per cent., evidently the value of this form of treatment had not impressed itself very seriously on the minds of our hospital physicians. There is no doubt that success has followed the use of both sera and vaccines in a large number of cases, but it seems likely that we have heard more of the successes than of the failures. In a recent book on "Serums, Vaccines, and Toxines" (1910) it is suggested that in case of failure with one brand of serum another brand may be successful. It is evident that in streptococcus disease we should not use antistaphylococcal serum, yet apparently this has sometimes been done. Some temporary benefit has apparently followed the use of His's serum. But in my nine hospital cases both sera and vaccines were used ineffectually as to ultimate result. Nor was unguentum Credé, used in one instance, successful.

In any event, the indications are for rest, good and abundant

food, the liberation of pus when practicable, and the use of sulphate of quinine and perhaps iodine internally, and supporting treatment generally.

In the five cases I have reported as improved or cured it appears from the records that the success was due to the use of expectant treatment. In them no specific was given, but, so far as I was able to judge, each individual symptom was treated as it manifested itself. And yet, in spite of all I have said, and all that I found from the study of hospital cases, I have been impressed with the idea that Wright often gets remarkable results, and that bacterial therapy has probably come to stay. At the same time it remains for us to discover not only the precise cases in which it is applicable, but more particularly the character of the bacterins or sera to be used and the technique required to produce successful results. Meanwhile the study of my cases illustrates that a cure is possible in 5 per cent. by the use of symptomatic measures.

Note.—Since writing the above, my attention has been called to an article on bacterial therapy (Synnott, *Medical Record,* 1911, LXXIX. 985) which the author regards as contraindicated in malignant endocarditis and puerperal septicemia as soon as the invading bacteria have gained access to the blood stream, causing systematic infection. At best, it is thought to be of problematical value in these cases.

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ERRATA.

The attention of the reader is called to the following errors and omissions:

On page 11, for intra-muscular read intermuscular.

On pages 15, 93, and 162 for Cushing read Cushny.

On pages 15, 93, and 162 for Edwards read Edmunds.

On page 44, in description of Fig. 19, for 10/12 read 12/10.

On page 45, for 1 1/2 seconds read 1/10 to 1/20 of a second.

On page 67, the ⊥ ⊥ alluded to in the description of Fig. 39 have been omitted in the illustration, but the extra-auricular systoles are plainly visible in the electrographic tracing.

On page 88, for Fig. 1 read Fig. 51.

On page 95, for Fig. 6 read Fig. 57.

On page 115, note omission of the illustration alluded to.

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Critical Notices of Cardiovascular Diseases by Thomas E. Satterthwaite, A.B., M.D., LL.D., Sc.D. New York: Lemcke & Buecher, 1913.

This work presents in attractive form the results of the labor of many diligent and capable men. Among the anatomists, Keith, Flack, Tawara; of the physiologists, Gaskell, Einthoven, Lewis, Cushny, and from the clinicans, Mackenzie, Hering, Janowski, and not omitting the author, should be cited as having made substantial contributions to the better understanding of a class of diseases second only to tuberculosis in their fatality. The results of laboratory and clinical observations of this last decade have been recorded in numerous journals; here we find all that is of importance presented in attractive form, so that this book is a fitting supplement to the "Diseases of the Heart and Aorta," published by the author in 1905. The value of the volume is enhanced by the large number of illustrations of instruments and tracings produced by them, and their use is fully explained. Although much doubt as to the interpretation of graphic pulse tracings must exist until the normal is sharply defined and the abnormal classified, yet for permanency and comparative study these methods constitute a real advance and will likely lead to more accurate knowledge. Of the chapters which appeal more strongly to the physician, those on the high frequency currents in arteriosclerosis, uses of carbon dioxide, and the cardiovascular thromboses are unusually helpful. The mobility and malpositions of the heart can nowhere else be found so succinctly presented as here. With the author's practical classification of the arrhythmias, myocardial diseases, and malignant endocarditis, not only the clearing up of our knowledge but distinct advance in the management of these conditions has been accomplished. From the study of this work not only the general physician but as well those working in the narrower field will acquire valuable information, critically presented by a brilliant exponent of diagnosis and treatment.—*Medical Record,* April 19, 1913.

The rapid advances made within the last few years in cardiology render it necessary that the practitioner should revise knowledge acquired less recently in order that he may keep pace with these. Dr. Satterthwaite's book is, therefore, most opportune, as it supplies in handy form an account of all the latest additions to our knowledge in his department of medicine. Modern teaching on the anatomy and physiology of the heart is fully considered. Sphygmomanometry is dealt with at considerable length. Graphic methods, as applied to the diagnosis of cardiac affections, are clearly described. The electrocardiograph and its use are very well explained, while some of the newer instruments used in the study of heart disease, such as the viscometer, are described and illustrated. Chapters are given on cardiac arhythmia, the use of high frequency currents in arteriosclerosis, the uses of carbon dioxide in heart disease, the mobility and malpositions of the heart, cardiovascular thrombosis, myocardial disease, and malignant endocarditis. The author's classification of diseases of the myocardium is novel, and has much to recommend it; while his hints on treatment are invaluable. The volume is well illustrated, and deserves to be widely studied by the profession, as it contains in short and accessible form a most readable account of recent advances in the domain of heart

affections. The writer is a man of great experience, and he expresses opinions based on first-hand knowledge. Those of our readers who are interested in this fascinating subject will find an hour or two spent in the study of Dr. Satterthwaite's book a most profitable exercise. To compress so much information within the compass of 160 pages must have been no easy task, but the author has accomplished it with credit to himself, as anyone who takes the trouble to read what the author has set down will be bound to admit. Personally we have studied this delightful volume with much pleasure and still greater profit.—*London Medical Times,* May 31, 1913.

A series of monographs in book-form issued as an addendum to the author's work on Diseases of the Heart and Aorta. These monographs have been revised as they include all of the recent advances in the anatomy, physiology, pathology, diagnosis and treatment of cardiovascular diseases.

All needlessly technical terms have been avoided, a task which ought to make this book still more attractive to the general practitioner.—Dupaquier, *New Orleans Medical and Surgical Journal.*

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